PROSPECTIVE CLINICAL STUDY ON HELLP SYNDROME

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CERTIFICATE

This is to certify that the Dissertation titled **"PROSPECTIVE CLINICAL STUDY ON HELLP SYNDROME"** is the bonafide original work of **Dr.N.S.SARADHA** in partial fulfillment of the requirements for **M.D Branch-II (Obstetrics and Gynecology)** Examination of The Tamilnadu Dr.M.G.R.Medical University to be held in March 2007.

The period of this study was from April 2005 to March 2006.

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I **Dr.N.S.SARADHA** Solemnly declare that the dissertation titled **"PROSPECTIVE CLINICAL STUDY ON HELLP SYNDROME"** is a bonafide work done by me at Govt.RSRM Lying-in Hospital, Stanley Medical College, Chennai-600 001,during April 2005 to March 2006,under the guidance and supervision of **Prof.Dr.B.RUPA.M.D.,D.G.O.,**

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CONTENTS

INTRODUCTION

INTRODUCTION

The clinical triad of hypertension, proteinuria and non dependent edema is a well recognised syndrome of the second half of pregnancy known as preeclampsia, although its etiology remains uncertain. Patient cases that were variants of pre-eclampsia-eclampsia with atypical signs and symptoms or very complicated courses of this disease process have been reported since the end of 19th century. Many of these descriptions of pregnancies complicated by an unique presentation of preeclampsia-eclampsia are representation of the condition now identified as **HELLP Syndrome**. The acronym was first suggested by **L. Weinstein in 1982** to describe the presence of **Hemolysis (H), elevated liver enzymes (EL)** as evidence of hepatic dysfunction and thrombocytopenia as evidence of **Low platelets (LP)** in a women considered to have preeclampsia-eclampsia.

Numerous investigations have been undertaken since 1982 to pursue a better understanding and to enlighten the treatment of potentially life-threatening entity. Recent investigators have provided evidence some cases of HELLP syndrome represent a vasculopathy mediated by an abnormal concentration of vascular growth factors. However until the underlying etiology for preeclampsia is better defined and testing for such factors is commonplace, controversies in the diagnosis and management of HELLP syndrome will persist as its numerous clinical findings will lead to varied impressions of severity and to varied thresholds for intervention. Although controversy surrounds almost every aspect of HELLP Syndrome, the balance of current opinion is that it does exist as a distrinct entity.

HELLP syndrome has been described as first appearing from the mid second trimester of pregnancy until several days postpartum.

AIM OF STUDY

AIM OF THE STUDY

1. To determine the occurrence and the course of HELLP syndrome in all preeclamptic-eclamptic cases, and its clinical presentation, subsequent management and maternal and perinatal outcome.

2. To asses the extent of disease process in the mother and its effect on the fetus by doing necessary investigations.

3. To time the delivery so that the mother and the fetus best tolerate the delivery process and to give the fetus the best chance of extra uterine existence.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Historical aspects of HELLP Syndrome

The preeclamptic patient with the constellation of hemolysis, hepatic dysfunction, and low platelets has been described in the literature for decades with early accurate descriptions by Prichard et al (1954) and Chesley. It was not until 1982, however, when **Weinstein Coined the acronym HELLP Syndrome** (hemolysis, elevated liver enzymes and low platelets) that clinicians could more easily recognise and discuss this group of patients with remarkable hepatic involvement by severe preeclampsia. Since Weinstein's publication, numerous reports of this syndrome have been published.

Reviewing literature reveals the confusion and considerable controversy concerning the existence, definition, incidence, diagnosis and management of this syndrome. Goodlin considered it an early form of severe preeclampsia and labelled as a great imitator, impending gestosis, EPH (edema, proteinuria, hypertension), Gestosis type B and extended toxemia syndrome. Weinstein considered it an unique variant of preeclampsia, while Mackenna and Colleagues considered it as misdiagnosed preeclampsia.

PATHOPHYSIOLOGY

HELLP SYNDROME:

Possible Pathophysiology

Inciting Agent(s): Sudden Large Volume Fetal/Decidual Cell Traffic? Vasospasm? Vascular Repair Deficiency? , Unknown(s)?

Vascular-Endothelial Dysfunction

Platelet Aggregation – Consumption

Fibrin Activation – Consumption

Selective Organ(s) Ischemia-Insufficiency

Diverse Symptomatology

It has been accepted that theory of disequilibrium in prostanoid metabolism in its etiology.

RED BLOOD CELL MORPHOLOGY

Fragmented red blood cells are present in women with HELLP Syndrome, but the amount of fragmentation often does not correlate with the degree of multi organ involvement that is reflected by other laboratory tests.

PLATELETS

The normal life span of a platelet is 8-10 days. In pregnancies complicated by preeclampsia, the life span of a platelet is reduced to 3-5 days. A further reduction in platelet life span and structural integrity is observed in women with HELLP syndrome, leading to further platelet aggregation and destruction. The altered platelet membrane in

HELLP syndrome releases arachidonic acid and other vasoactive amines, causing further vasoconstriction and vasospasm and accelerating platelet aggregation and destruction. Platelet activating factor (PAF), a potent activator of platelet aggregation, appears not to be altered by preeclampsia. One of the serum inhibitors of PAF is reduced. Platelet activation and alterations in plasminogen activation are involved in the pathogenesis of this disorder. Beta-thrombomodulin is a platelet specific protein whose levels become elevated. Early in the HELLP syndrome, there is probably a procoagulatory imbalance in the placental microcirculation.

RENAL COMPROMISE

A small percentage of HELLP syndrome patients develop significant renal compromise. The disease spectrum ranges from transient elevations of serum creatinine concentrations to acute renal failure with temporary and reversible acute tubular necrosis (ATN) to permanent and irreversible renal failure with cortical necrosis.

Most commonly HELLP Syndrome involves smaller terminal arterioles yielding a process with characteristic histological features. The classic hepatic lesion associated with HELLP syndrome is periportal or focal parenchymal necrosis in which hyaline deposits of fibrin like material can be seen in the Sinusoids. Less frequently larger vessel disease can impact wider vascular distributions in the liver with more catastrophic outcomes such as hepatic infarction and subcapsular hematoma. This large vessel disease is more readily visible by imaging studies such as MRI and CT scanning. The most common site of hepatic rupture is the anterior superior aspect of the right lobe of the liver, and liver ruptures have been reported as early as 16 weeks gestation to 3 days post-partum.

Pathogenesis of HELLP syndrome is associated with factor V R 500 Q mutation (Brenner et al. 1996) Activated Protein C resistance resulting from mutation in coagulation factor V has recently emerged as the leading cause of thrombosis.

Patients with pure preeclampsia and HELLP Syndrome will have significantly higher level of serum C-erb B-2 encoded oncoprotein fragment P_{105} (Meden et al. 1997)

Lower beta B-subunit Inhibin production in extra villous trophoblast cells in HELLP syndrome demonstrates that this subunit might have an important role in the pathogenesis of HELLP syndrome.

INCIDENCE

Based on 454 pregnancies with HELLP syndrome managed at the University of Mississippi Medical Center between 1980 and 1991 and 442 pregnancies cared for at the University of Tennessee-Memphis between 1977 and 1992, approximately one third of patients will have their first manifestation of HELLP syndrome diagnosed postpartum, of the two thirds of women who are first diagnosed with HELLP syndrome antepartum, 10% will be identified before 27 weeks, 20% in pregnancies beyond 37 weeks, and the majority 70% occurring between 27 and 37 weeks gestation. The earliest recorded case of HELLP Syndrome in Mississippi was at 20 weeks, although a diagnosis before 25 weeks is infrequent.

The incidence is highest among older, white and multiparous patients. Moreover the incidence is higher in preeclamptic patients with delayed diagnosis and/or delayed delivery.

CLINICAL PRESENTATION

Patients with HELLP syndrome may present with various signs and symptoms, none of which are diagnostic and all of which be found in patients with severe preeclampsia-eclampsia without HELLP syndrome.

Prodromal Symptoms include (Portis et al., 1997)

1. Weakness and fatigue (90%)
2. Right upper quadrant and/or epigastric pain (90%)
3. Nausea and Vomiting (50%)
4. Headache
5. Change in vision
6. Increased tendency to bleed from minor trauma
7. Jaundice

8. Diarrhoea

9. Shoulder or neck pain

Signs

1. Significant weight gain with generalised edema (55%)

2. Proteinuria ≥ 2+ (85%)

3. Diastolic Blood Pressure ≥ 100 mm Hg (69%)

Sibai et al (1990) noted that the commonest symptom was epigastric and/or right upper quadrant pain. In Weinstein reports (1982/1985) nausea or vomiting and epigastric pain were the most common symptoms. Although the contribution of right upper quadrant or epigastric pain to the risk status of a pregnant patient is difficult to quantify, it can be used to assess whether the patient is at high risk for development of HELLP syndrome.

A diurnal pattern exists in the clinical symptoms of HELLP syndrome that is characterised by an exacerbation during the night and recovery during the day. There is considerable delay between the onset of symptoms and the fulfillment of diagnostic laboratory criteria (Koenen SV et al 2006).

It is important to appreciate that severe hypertension (systolic blood pressure ≥ 160 mm Hg, diastolic blood pressure ≥ 110mm Hg) is not a constant or even a frequent finding in HELLP syndrome (Sibai et al 1986). Esan et al (1997) reported that HELLP syndrome can occur after a normal delivery in a woman whose blood pressure has remained normal throughout the antenatal period.

Jaundice is a rare complication and hyperbilirubinemia may result from a combination of hemolysis and liver cell necrosis. However it is unusual for icterus to be clinically apparent.

Conversely, hemoglobinuria is a more common finding when complicated by disseminated intravascular coagulation.

Donaldson (1978) reported that some may experience visual disturbances.

Neurological affection can also result. The risk of opportunistic infections may be increased in patients with HELLP syndrome, because of generalised (Both B&T Cell) immunosuppression and profound decrease in monocyte phagocytic and bactericidal activity (Cunningham et al. 1993).

Maternal ascitis is frequently found at Caesarean delivery in 65% of patients with HELLP syndrome (Woods et al 1992).

LABORATORY DIAGNOSIS

The diagnosis of HELLP Syndrome is based on laboratory evidence of microangiopathic hemolytic anemia, hepatic dysfunction and thrombocytopenia in a patient suspected to have preeclampsia. In a patient with HELLP syndrome, a peripheral blood smear often will have evidence of schistocytes, burr cells and helmet cells which reflect damaged erythrocytes. Increases in Lactic dehydrogenase (LDH) levels and decreases in serum Haptaglobin levels are sensitive early markers of HELLP syndrome. Thrombocytopenia is the principal and earliest coagulation abnormality that is present in all women with HELLP syndrome.

Liver dysfunction is reflected by variably elevated serum concentration of aspartate amino transaminase (AST), alanine aminotransaminase (ALT) and LDH. Indirect levels of bilirubin usually are minimally elevated except in patients with advanced severe disease. Raised total Lactate dehydrogenase isoenzyme is usually reflected in elevations of isoenzymes 5 (LDH 5 liver).

HELLP syndrome patients will have high plasma fibronectin and D-dimer values, lower Antithrombin III and protein C activity (Paternoster et al. 1995).

HELLP syndrome should be taken into account in the case of unexplained elevated levels of MSHCG and MSAFP in the second trimester especially in the rare event of combined elevation of both markers (Morssink-LP 1997).

Iioka (1996) reported that increased level of Human Hepatocyte growth factor may be useful in the early detection of HELLP syndrome.

Significant over expression of Serum Amyloid A (SAA) in HELLP syndrome

patients that could function as markers for the HELLP syndrome (Heitner JC et al 2006).

A single A > G nucleotide substitution at position - 670 in the maternal but not neonatal TNFRSF6 gene coding for Fas is associated with a higher risk for HELLP syndrome (Sziller et al 2006). Significantly there will be a decreased expression of Pro apoptotic proteins BNip3 and Nix in the placenta of HELLP syndrome patients (Stepan H. et al 2005). O Rh-negative had HELLP syndrome associated with an increase in risk by a factor of 3.1 (Sezik M & Coworkers 2002).

CLASSIFICATION

A three-class system was formulated because the maternal platelet count and the serum LDH concentration appear to best reflect the severity of the disease process as it worsens and the rapidity of recovery from HELLP syndrome. Women with Class I HELLP syndrome have a maternal platelet nadir of $\leq$ 50,000 /μL in addition to laboratory evidence of microangiopathic hemolytic anemia and altered liver enzymes. Patients with class II HELLP syndrome have a platelet nadir of >50,000 but $\leq$ 1,00,000/μL, and those with class III disease have a platelet count of > 1,00,000 but $\leq$ 1,50,000/μL

HELLP SYNDROME

Classification Systems

Mississippi 3-class (Martin et al)	Tennessee (Memphis)
THROMBOCYTOPENIA	**COMPLETE**
CLASS 1 : ≤ 50,000 / μL Platelets	< 1,00,000/μ L Platelets
CLASS 2 : > 50,000 - ≤ 1,00,000/ μ L	LDH ≥ 600 IU/L
CLASS 3 : > 1,00,000 - ≤ 1,50,000/ μ L	AST ≥ 70 IU/L
HAEMOLYSIS + HEPATIC DYSFUNCTION	**INCOMPLETE**
LDH ≥ 600 IU/L	Only one or two of above present
AST and/or ALT ≥ 40 IU/L	
All must be present to qualify...	

Other classifications of HELLP Syndrome

	Platelet Count	AST	LDH
Sibai et al [11]	< 1,00,000/mm^3	≥ 70 IU/L	≥ 600 IU/L
Van Pampus[13]	< 1,00,000/mm^3	> 50 IU/L	>600 IU/L
Visser and Wallenburg[14]	< 1,00,000mm^3	> 30 IU/L	*

DIFFERENTIAL DIAGNOSIS OF PRE ECLAMPSIA - HELLP SYNDROME

- THROMBOTIC MICROANGIOPATHIES

  Thrombotic Thrombocytopenic purpura - Hemolytic Uremic Syndrome Sepsis-Induced or Drug Induced Microangiopathic Hemolytic Anemia.

- FIBRINOGEN CONSUMPTIVE DISORDERS - DIC

  AFLP-Acute Fatty Liver of Pregnancy

  Severe Hypovolemia/Hemorrhage (Abruption/Amniotic Fluid Embolism)

  Sepsis Induced.

- CONNECTIVE TISSUE DISORDERS

SLE - Systemic Lupus Erythematosis

- PRIMARY RENAL DISEASE
- MISCELLANEOUS

Viral Hepatitis	Kidney Stones
Hyperemesis gravidarum	Gastroenteritis
Gallbladder disease	Diabetes Insipidus
Peptic Ulcer	Hepatic encephalopathy
Appendicitis	Cholestasis of Pregnancy

MANAGEMENT

Reported modalities used to treat or reverse HELLP Syndrome (Sibai et al 1990)

I PLASMA VOLUME EXPANSION

- Bed rest
- Crystalloids
- Albumin 5% to 25%

II ANTITHROMBOTIC AGENTS

- Low dose aspirin
- Dipyridamole
- Heparin
- Antithrombin III
- Prostacyclin infusions
- Thromboxane Synthetase inhibitor (Dazoxiban)

III IMMUNOSUPPRESSIVE AGENTS

- Steroids

IV MISCELLANEOUS

- Fresh frozen Plasma infusions
- Exchange Plasmapharesis
- Dialysis

Intravenous immunoglobulins might be an attractive alternative treatment in persistently severe HELLP Syndrome (O-Pourrat et al 1992).

Another intervention to interrupt or ameliorate the clinical course of HELLP Syndrome includes the administration of nitric oxide.

Steroids and the HELLP Syndrome

"DEXAMETHASONE RESCUE" for HELLP SYNDROME

ANTEPARTUM: 10 mg IV q 12 h

1. Whenever < 100,000/ μL Platelets

2. If Platelets 100,000 - 150,000/μL AND

Eclampsia	Severe Hypertension
Epigastric pain	"Fulminant Disease"

POSTPARTUM: 10 mg IV q 12 h x 2, then 5 mg IV q 12h x 2 individualised

1. Whenever antepartum steroids given to avoid rebound

2. Stop regimen after recovery evident (platelets > 100,000/ μL and LDH is trending downward and patients underlying preeclampsia / eclampsia is ameliorating

The mechanism of action is unknown but appears to alter the final steps in endothelial cell disruption Isler et al demonstrated intravenous dosing was superior to intramuscular dosing for several outcome variables including improving urine output and greater improvement in Laboratory values.

High-Dose Glucocorticoid Therapy for severe HELLP Syndrome

For most patients with HELLP syndrome, 10 mg intravenous dexamethasone every 6 hours for 2 doses followed by 6 mg intravenous dexamethosone every 6 hours for 2 additional doses.

For select patients at high risk, including those with profound thrombocytopenia (<20,000/ mm^3) or with central nervous system dysfunction (i.e. blindness, paralysis), 20 mg intravenous dexamethasone every 6 hours for upto 4 doses.

The duration of action of this medication is limited and patients may experience a worsening of their laboratory studies 48 to 72 hours after dosing with glucocorticoids. We term this as Rebound phenomenon. Steroid treatment, therefore, is not curative but may create a "Window of opportunity" for intervention before the maternal condition may again deteriorate. Because glucocorticoids do not appear to alter the underlying pathophysiology, delivery remains the only definitive therapy.

MATERNAL MORBIDITY AND MORTALITY

Maternal morbidity has been reported to be as high as 24% in one series of patients, but should be infrequent in an optimal medical and obstetric environment. The maternal death does not occur by a common pathway, but instead by a variety of mechanisms. In a review of 34 HELLP syndrome related mortalities, the authors discovered that the presenting symptom in 90% of patients who died was nausea-vomiting and right upper quadrant pain, the mean gestational age was 31 weeks, death occurred by a variety of pathologic processes, including sepsis, shock hemorrhage, intra cerebral bleeding and cardiopulmonary failure. Approximately one in six (16%) maternal deaths was attributed to hepatic complications. A large percentage of maternal deaths attributed to central nervous system catastrophic events. The most important biochemical marker for maternal mortality is bilirubin levels. Maternal mortality was statistically higher in cases with jaundice (Demir SC et al 2006).

The diverse etiology of maternal mortality in HELLP syndrome is

Cardiopulmonary Failure

Coagulopathy

Intracerebral Hemorrhage/stroke

Hepatic Rupture

Post-Cesarean Shock

Multi organ Failure

Miscellaneous Co-morbidities

Serious maternal morbidity is observed in patients with HELLP syndrome.

From Sibai et al (2003)

1.	Disseminated intravascular coagulopathy	21%
2.	Abruptio placentae	16%
3.	Acute renal failure	8%
4.	Severe ascitis	8%
5.	Pulmonary edema	6%
6.	Pleural effusions	6%
7.	Cerebral edema	1%
8.	Retinal detachment	1%
9.	Laryngeal edema	1%
10.	Subcapsular liver hematoma	1%
11.	Acute respiratory distress syndrome	1%

Patients with Class 1 disease are at the highest risk for associated morbidity, with proportionately less observed in women with Class 2 HELLP Syndrome.

Associated DIC is an important aggravating factor, often leading to deterioration of the maternal status, Van Dam et al (1989) suggested a semi quantitative DIC scoring system introduced by Hellgren et al (1984). This DIC score is based on platelet count less than 100 x 10 9/7, Prothrombin time < 70%, antithrombin III activity < 80%, fibrin degradation products over 40 mg/L and fibrinogen < 300 mg/dl. Three or more pathologic tests were considered as manifest DIC and two as suspected DIC. DIC score may be a sensitive index for detecting deteriorating maternal condition in HELLP syndrome and its use could reduce maternal morbidity and mortality from DIC.

A rare but interesting complication of HELLP Syndrome is transient Diabetes Insipidus (Mabie & Sibai 16 1990). It is characterised by a resistance to arginine vasopressin mediated by excessive vasopressinase. It is postulated that elevated circulating vasopressinase may result from impaired hepatic metabolism of the enzyme.

The best prophylaxis against development of life threatening complications is early diagnosis and termination of pregnancy after stabilisation of the maternal condition, consisting of magnesium sulphate infusion, antihypertensive treatment with dihydralazine or calcium antagonists, steroids etc. As prophylaxis against postpartal worsening of HELLP syndrome, curettage of the uterus and continuation of the treatment with antihypertensives and dexamethasone have been recommended.

PERINATAL MORBIDITY AND MORTALITY

Perinatal morbidity and mortality are increased in women with HELLP syndrome primarily because of prematurity. No specific neonatal pathology due to maternal HELLP syndrome was found.

Perinatal mortality 33% (Sibai et al 1986, Eeltink et al 1993) reported

- Small for gestational age 44%
- Perinatal asphyxia 21.6%
- Neonatal respiratory distress 43.2%

- Hyperbilirubinemia 44.7%
- Persistent ductus arteriosus 16.2%
- Thrombocytopenia 34%
- Hypoglycemia 16.2%

CAUSES FOR PERINATAL DEATH

1. Abruptio placentae

2. Intrauterine asphyxia

3. Prematurity

The combination of HELLP syndrome and eclampsia results in a greater number of preterm infants with lower birth weights and higher mortalities than eclampsia alone.

IMMUNOLOGY OF HELLP SYNDROME

Pre eclampsia has been considered for a number of years by many investigators to result at least in part, from disordered immunologic processes. The studies that support this belief, list the increased incidence in primigravida, the increased risk in pregnancies with an increased volume of trophoblastic tissue, pregnancy with a new partner, previous use of barrier contraceptive and pregnancy after oocyte donation. Since HELLP syndrome appears to be atypical form of preeclampsia, it too could result from disordered immunity.

Increased plasma levels of anaphylotoxins C3a and C5a have been demonstrated in patients with preeclampsia / HELLP syndrome. Depressions of both T-cell and B-cell potential and impaired monocyte handling of intracellular pathogens have been reported in pregnancies complicated by HELLP syndrome. This immune dysfunction preceded the laboratory diagnosis of preeclampsia by 7-14 days.

Haeger et al (1996) suggested that inflammatory mechanisms may participate in the pathophysiology of severe preeclampsia since increased release of Tumor necrosis factor alpha (TNFα) and interleukin 6 in women with HELLP Syndrome.

Dudley et al (1996) supported the hypothesis that the regulation of IL-12 production and metabolism is abnormal in women with preeclampsia and HELLP Syndrome, perhaps contributing to the immunologic alterations characteristics of these disorders.

Antiphospholipid antibodies may play a role in the pathogenesis of HELLP syndrome (Nagayama et al 1997).

PATIENT TREATMENT: The 12-Step Approach

Successful management of pregnancy complicated by HELLP Syndrome requires early recognition and the institution of the appropriate therapy.

1. ANTICIPATE AND MAKE THE DIAGNOSIS

Once the diagnosis of HELLP Syndrome is suspected, appropriate laboratory testing is indicated. In its early phases, HELLP syndrome can exhibit only modest increases in LDH, AST and ALT levels and mild thrombocytopenia (Class 3). The patient with evidence of preeclampsia and right upper quadrant pain and nausea must be seriously evaluated to rule out HELLP syndrome. Timely diagnosis facilitates the best possible outcome for a mother and her unborn child.

HELLP SYNDROME: Risk Factors for

Significant Maternal Morbidity

• **LABORATORY**	• **CLINICAL**
Platelets < 50,000/ μ L	Epigastric pain
LDH > 1400 IU/L	Nausea
AST > 150 IU/L	Vomiting
ALT > 100 IU/L	Eclampsia
Uric Acid > 7.8 mg/dl	Severe Hypertension
CPK > 200 IU/L	Placental Abruption
Creatinine > 1.0	

2. ASSESS THE MATERNAL CONDITION

The basic laboratory screening for the patient with suspected HELLP syndrome is a complete blood count with platelet levels, urinalysis, serum creatinine, LDH, uric acid, indirect and total bilirubin levels, and AST/ALT. Tests for prothrombin time, partial thromboplastin time, fibrinogen and fibrin split products are reserved for those women with a platelet count much below 1,00,000/ μL. They are particularly useful in women with platelet counts of 50,000 or less.

Serial assessments of the platelet count, LDH, and liver enzymes are reported usually every 12-24 hrs or more frequently if clinically indicated.

3. ASSESS THE FETAL CONDITION: DELIVER SOONER OR LATER?

HELLP syndrome is an atypical variant of severe preeclampsia and, as such, the only definitive treatment is delivery and removal of chorionic villi and the cytotoxic factor(s) that it produces. The timing of delivery depends on a number of factors, including the severity of the maternal condition, fetal condition and placental reserve, and

the gestational age. The facilities available to care for this high risk mother and often preterm infant also will influence the location of the delivery, with transport often necessary before delivery to a hospital equipped to manage these at risk mothers and babies.

A National Institute of Health consensus panel in 1994 recommended that all patients with pregnancies between 24 and 34 weeks gestation at risk for preterm delivery be considered candidates for corticosteroid therapy to enhance fetal lung maturation/neonatal pulmonary function even if delivery might not be postponed the ideal 24-48 hour period. ACOG committee on Obstetrical practice has now adopted these recommendations.

The longest interval we have used corticosteroids to postpone delivery in very preterm (24 weeks) patients with HELLP syndrome is 19 days.

4. CONTROL BLOOD PRESSURE

5. PREVENT SEIZURES WITH MAGNESIUM SULPHATE

It is recommended that all HELLP syndrome patients receive intravenously infused $MgSO_4$ given as a 4-6 gm intravenous bolus followed by a constant infusion rate of 1.5 - 4 gm/hr individualized to the patient. Continuation of the infusion 48 hrs or more into the puerperium occasionally is needed until evidence of recovery from HELLP syndrome is apparent.

6. MANAGE FLUID AND ELECTROLYTES

7. EXERCISE JUDICIOUS HEMOTHERAPY

8. MANAGE LABOUR AND DELIVERY

Current hospital treatment of the patient with HELLP syndrome in United States includes careful assessment of the maternal and fetal status, with delivery effected soon thereafter.

The presence of this syndrome is not an indication for immediate delivery by caesarean section; such an approach might prove detrimental for both mother and fetus.

Cervical status and inducibility are important considerations when determining the likelihood of successful vaginal delivery in patient with HELLP syndrome.

Management protocol for the HELLP Syndrome patient requiring caesarean section delivery

1. General anesthesia

2. Ten units of platelets prior to surgery if platelet counts < 50,000/cu mm.

3. Vertical skin incision rather than a Pfannenstiel incision to minimise blood loss.

   Briggs et al concluded that for women with antepartum HELLP syndrome delivered by caesarean section, the frequency of wound complications is not influenced by type of skin incision or time of skin closure (Primary or delayed).

4. Spontaneous placental rather than manual extraction at caesarean is associated with a decreased blood loss, and less postpartum endometritis.

5. In situ rather than exteriorization for uterine repair is associated with less uterine and adnexal trauma.

6. Bladder flap (vesicouterine peritoneum) should be left open.

7. Subfascial drain (24 to 48 hours).

8. Subcutaneous drain may be considered if the skin is approximated.

9. Subcutaneous tissues should be approximated with sutures as evidenced by randomized trials and meta analysis for skin closure at caesarean delivery.

10. Post operative transfusions as needed.

11. Intensive monitoring for 48 hrs postpartum.

ANAESTHESIA

- Maternal analgesia can be provided by intermittent infusion of butorphanol or

meperidine with promethazine.

- Local infiltration 1% xylocaine for carefully controlled and skillfully executed non operative vaginal delivery.

- Epidural anesthesia and pudendal block is contraindicated.

- General anesthesia is the method of choice for caesarean delivery.

- When coagulopathy is evident before surgery, intradural anaesthesia (low doses of Bupivacaine and fentanyl) is a safe option provided hemodynamic stability is assured (Blasi et al 1997).

9. OPTIMIZE PERINATAL CARE

10. INTENSIVELY TREAT THE POSTPARTUM PATIENT

- The diagnostic criteria for HELLP syndrome may develop antepartum or postpartum. Sebai and associates revealed that 70% had evidence of the syndrome antepartum and 30% developed the criteria postpartum.

- In the postpartum group, the time of onset of the manifestations ranged from a few hours to 7 days, with the majority developing within 48 hours after delivery.

- HELLP syndrome may be diagnosed postpartum following 1 of 3 clinical scenarios:

1. Worsening of antepartum severe preeclampsia with delivery not yet altering the time course of the disease.

2. New onset of severe preeclampsia postpartum.

3. Rebound deterioration of a patient with antepartum HELLP syndrome after exposure to corticosteroid antepartum.

- Patients in this group are at increased risk for the development of pulmonary edema and acute renal failure.

- The goals of therapy postpartum differ compared with antepartum and are aimed solely at improving the maternal status.

- Management of seizure prophylaxis is similar to the antepartum patient with HELLP syndrome, including the need for $MgSO_4$.

- Hypertension control may be more aggressive, because there is no longer concern about compromising the uteroplacental circulation in the postpartum patient.

- Martin and coworkers recommended the trial of plasma exchange with fresh frozen plasma be considered in HELLP syndrome that persists past 72 hours from delivery and in which there is evidence of a life threatening microangiopathy.

11. REMAIN ALERT TO THE DEVELOPMENT OF MULTIPLE ORGAN SYSTEM FAILURE

12. COUNSEL ABOUT FUTURE PREGNANCIES

During the postpartum recovery period after a pregnancy with HELLP syndrome or at a later time remote from the index pregnancy, patients often ask for guidance about the recurrence risk for hypertension in general and HELLP syndrome specially in future pregnancies.

Currently there is no preventive therapy for recurrent HELLP syndrome. Birth weight and gestational age are the most important factors in predicting the course of a subsequent pregnancy.

SUBSEQUENT PREGNANCY COMPLICATIONS: SIBAI ET AL (1995)

1.	Pre-eclampsia	19%
2.	Preterm delivery	21%
3.	IUGR	12%
4.	Abruptio placentae	2%
5.	Perinatal death	4%
6.	HELLP Syndrome	3-5%
7.	Chronic Hypertension	4%

Recurrence Risk for HELLP Syndrome or Preeclampsia

	HELLP Syndrome	**Pre eclampsia**
Sullivan et al	19 – 27%	23 - 43%
Lie 1998	-	13%
Van Pampus et al	2%	16%
Chames et al	6%	55%

Beinder et al 1996 reported that recurrence of HELLP Syndrome in four consecutive pregnancies in a patient.

Infants born to pre eclamptic mothers who develop HELLP Syndrome have an increased need for resuscitation at delivery and a higher incidence of postnatal cardio pulmonary instability (Raval et al 1997).

Sibai et al (1995) reported that there is no evidence that oral contraceptives should be contraindicated after HELLP syndrome.

MATERIALSAND METHODS

MATERIALS AND METHODS

Analysis of 200 cases of preeclampsia - eclampsia during the year 2005-2006 in Government R.S.R.M. Hospital, Chennai to determine the occurrence and course of HELLP Syndrome in order to make a timely intervention and to render optimal patient treatment, a better maternal and perinatal outcome.

The patients were divided into 3 groups

1. Group of Mild Preeclampsia
2. Group of Severe Preeclampsia
3. Group of Eclampsia

History regarding age, parity, gestational age, menstrual history and pervious illness were noted. A thorough general and other systemic examination were done with obstetric examination.

The observations done were

1. Weight of the patient
2. Albuminuria
3. Blood Pressure
4. Haemoglobin
5. Platelet count
6. Peripheral smear
7. Serum Bilirubin estimation
8. SGOT estimation
9. SGPT estimation
10. BT
11. CT

12. Serum fibrinogen

13. Sr. LDH estimation

14. Blood urea and serum uric acid estimation

15. Serum Creatinine estimation

16. Fundus examination

Group I

It consists of 68 cases of mild pre-eclampsia. It includes 40 cases of primi and 28 cases of multi, whose systolic blood pressure was between 130-150 mm. Hg and diastolic between 90-100 mm. Hg with proteinuria and pedal edema.

These patients were advised bed rest in left lateral position and given only sedation.

Group II

It consists of 100 cases of severe preeclampsia, includes 52 cases of primi and 48 cases of Multi. This group of patients had history of headache, giddiness and visual disturbances.

Urine showed proteinuria and there was Oedema over both legs in all cases. These patients were kept under strict bed rest, daily weight, daily urine for proteinuria and BP estimation were done. Since, these patients had systolic blood pressure between 150-200 mm. Hg and diastolic blood pressure between 110-130 mm.Hg, patients were given Tab. Alphamethyldopa, Tab. Nifedipine and Inj. Pethidine 100mg depending upon the blood pressure.

Daily urine output was measured and the patients were advised to lie on the left lateral position. The uterus was examined for tenderness and tenseness and growth.

Group III

It consists of 32 cases of Eclampsia, includes 24 cases of Antepartum Eclampsia,

(Primi 13 cases and 11 cases of Multi), 3 cases of intrapartum Eclampsia (1 primi and 2 multi) and 5 cases of postpartum eclampsia (3 cases of primi and 2 cases of multi).

All patients were kept in Eclampsia ward. The management was done by monitoring blood pressure every 2 hours, continuous bladder drainage, intake output chart was maintained along with intravenous fluids, antibiotics and $MgSO_4$ regime.

Intermittent suction was done and oxygen (O_2) kept ready for emergency.

METHOD OF COUNTING PLATELETS

Rapid work is necessary in order to prevent clumping of the thrombocytes. Rees & Eker diluting fluid drawn upto 1 mark in the red pipette. Blood from a freely bleeding puncture is drawn exactly to the 0.5 mark and finally the diluting fluid is quickly drawn to the 101 mark. This gives a blood dilution of 1:200. The blood and diluting fluids are immediately mixed by shaking for about 2 mts. The counting chamber is filled at once and 10 mts are allowed for the corpuscles to settle before counting is begun. The count is made with high power dry objective and with the 10x ocular in the manner described for counting erythrocytes. A central count of the thrombocytes should always be made at the same time with the same diluting fluid and exactly the same technique. Platelet count were also obtained from peripheral smear.

Platelets are disc shaped being 2-4 μm in diameter and 0.5 to 1mm in thickness. They have no nucleus and their cytoplasm has many azurophil granules which in blood films tend to be concentrated in the middle. The precursor for the platelet is megakaryocytes. The platelets are formed by fragmentation and detachment of delicate processes from cytoplasm of megakaryocyte. The normal platelet count varies from 140000 to 440000/mm^3. A count below 100000/mm^3 can be taken as thrombocytopenia.

PERIPHERAL SMEAR

Spreading the Film

Properly spread film is essential to accurate work.

1. The slides and cover glasses must be perfectly clean
2. Drop of blood must not be too large
3. The work must be done quickly

The blood is obtained from the finger tip. Take a small drop of blood on a clear slide about 3/4 inch from the end taking care that the slide does not touch the skin. Place the end of a second slide against the surface of the first an angle of 30-40° and draw it against the drop of blood, push the spreader slide back along the other. The blood will follow and then smears should be made. The film may be allowed to dry in the air. Leishman's stain is added, and after 2 mts double the quantity of distilled water are added to the stain and waited for 7-10 mts. Then the stain is washed in the tap water and slide dried in air and viewed under oil immersion.

The mature red corpuscle appears greenish yellow in unstained preparations and is roughly circular in shape and seen on edge as biconcave disc. Cells with reduced concentration of heamoglobin are called hypochromic which may be so extensive that only a narrow rim of haemoglobin is left around the periphery. There cells are called pessary cells.

Spherocyte are small darkly staining cells or ring. Burr cells are mature red cells which possess one or more spiky projecting on their periphery seen in microangiopathic haemolytic anaemia.

SGOT ESTIMATION

Sample Collection, Storage and Stability

Although serum is preferred, plasma can be used. Anticoagulants such as heparin and EDTA can be used. Blood samples may be collected any time, although morning samples are preferred.

Samples with any visible hemolysis are not acceptable. Samples are stable for a

week at 2-8° C and for one month at 10°C samples should be brought to room temperature prior to use.

Reagents

1. Aspartate/Buffer

1A. NADH/MDH/LDH

2. Alpha-Ketoglutarate

Preparation of working solutions

Allow the reagents to attain the room temperature.

Solution (1): Quantitatively transfer the contents of Via 1A into bottle 1. Mix until completely dissolved.

Solution (2): Dissolve the contents of bottle 2 with 14 ml of distilled water.

Preparation of daily working solutions

Allow the solutions (1) and (2) to attain the room temperature.

The daily working solution should be prepared freshly according to the need in the proportions given below:

Solution (1): 3.0 ml

Solution (2): 0.3 ml

Mix thoroughly, use within 8 hours of preparation - store at 2-8°C.

Storage and stability of Reagents

Expiry date of reagents stored at 2-8°C are

Solution (1): for 3 months

Solution (2): for 4 months

Procedure

The samples and the daily working solution should be at the room temperature prior to use. The following general system parameter are used.

Reaction Type	:	Kinetic
Wavelength	:	340 mm
Flowcell Temp	:	37°C
Delay Time	:	60 Sec
No. of Readings	:	4
Interval	:	30 Sec
Sample Volume	:	100 Ml
Reagent Volume	:	1.0 ml
Pathlength	:	1 cm
Factor	:	1749
Zero setting with	:	Distilled water

Procedure limitations

- Haemolysis of sample can produce a significant positive error as red cells contain large amounts of GOT (AST).
- Samples with very high GOT activity cause an excessive consumption of NADH, resulting in a very low initial absorbance. When this occurs, the assay should be repeated with a diluted sample.
- Linearity of the method is upto 400 IU/L for higher values, dilute the sample suitably with 0.9% saline and repeat the assay. Apply proper dilution factor to calculate the final results.

Normal Values

Upto 40 IU/L (37°C)

SGPT ESTIMATION

Sample

Although serum is preferred, plasma can also be used with anticoagulants such as heparin or EDTA.

Morning samples are preferred. Serum or plasma GPT determination should be carried out on the same day as far as possible.

Samples are stable for about 3 days when stored tightly capped at 2-8°C or for two weeks at - 10°C.

Avoid using haemolysed or grossly contaminated samples. The samples should be brought to room temperature prior to use.

Reagents

1. Alanine / Buffer

2. NADH/LDH

3. Alpha - Ketoglutarate

Preparation of working solutions, storage and stability of reagents, procedure & procedure limitation.

Same as that for SGOT.

Normal values

Upto 40 IU/L (37°C)

LDH ESTIMATION

Sample

Although serum is preferred, plasma can be used only if heparin or EDTA is used as an anticoagulant. Avoid using hemolysed and grossly contaminated samples.

Serum or plasma should be separated from the blood sample as early as possible.

Serum or plasma can be used stored at 2-8°C for one week.

Reagents

1. NADH

1A. Buffer

2. Pyruvate

Preparation of working solutions

Allow the reagents to attain the room temperature. The Ames Autopak LDH reagent kit contains three bottles/vials each of reagent 1 & 1A and one bottle of reagent 2.

Solution (1): Transfer a small amount of the contents of one bottle (1A) into one bottle (1). Mix gently to dissolve and transfer the solution back to bottle (1A). Rinse bottle (1) with solution in bottle (1A).

Reagent (2): Ready for use

Preparation of Daily working solutions

Allow the solution (1) and reagent (2) to attain the room temperature.

The daily working solution should be prepared fresh according to the need. Mix 10 volumes of solution (1) with 1 volume of reagent (2) to obtain daily working solution.

Storage and stability of the Reagents

Expiry date of reagents stored at 2-8°C is indicated on the box label.

Solution (1) is stable for six weeks at 2-8°C Reagent (2) is stable at 2-8°C until the end of expiry date on the table.

Procedure

The samples and the daily working solution should be brought to room temperature prior to use. The general system parameters are

Reaction Type	:	Kinetic
Wavelength	:	340mm
Flow cell temperature	:	37°C
Delay time	:	60 Sec
No. of Readings	:	4
Interval	:	30 Sec
Sample Volume	:	30 µl
Reagent Volume	:	1.0 ml
Pathlength	:	1 cm
Factor	:	5520
Zero setting with	:	Distilled water

Procedure limitations

Plasma can be used only if heparin or EDTA is used as an anticoagulant Citrate and oxalate interfere with the test and hence should not be used.

Do not use haemolysed samples as haemolysis may give falsely elevated results.

The method is linear upto 1000 IU/L. For higher values, dilute the sample suitably with 0.9% saline and repeat the assay. Apply proper dilution factor to calculate the final results.

Normal Values

Upto 600 IU/L (37°C)

SERUM BILIRUBIN ESTIMATION

Methods of detecting and estimating Bilirubin in serum are based on the formation of the purple compound when bilirubin reacts with the diazo reagent introduced by vanden Berg.

Reagents

1. Absolute method

2. Diazo reagent prepared freshly by adding 0.3 ml of solution B to 10 ml of solution A.

a. Solution A

Dissolve 1 gm of sulphuric acid in 15 ml concentrated HC1 and make it to i liter with distilled water.

b. Solution B

- 0.5% sodium nitrite in water prepared at frequent intervals.

- Prepare a solution containing 10 mgm/100ml of chloroform. It may be necessary to reflex the mixture gently to dissolve the bilirubin.

Technique

Wash 0.2 ml of serum into 5.4 ml of 0.9% saline. For values above 15 mgms/0.1 ml serum in 5.5 ml water may be taken serum 0.2, 5.6 ml divided into 4 parts of 1.4 ml each.

	Total Bilirubin		**Direct Bilirubin**	
	Test	**Control**	**Test**	**Control**
Dilute Screen	1.4 ml	1.4 ml	1.4 ml	1.4 ml
UDBA	-	0.35 ml	-	0.35 ml
Diazo reagent	0.35	-	0.35	-
Water	-	-	1.75	1.75

Methanol	1.75	1.75	-	-

Let it stand for 10 mts read against water blank at 530mμ photoelectric colorimeter.

Standardization

Mg/bilirubin per 100 ml 0-10-20-30-40

MI Standard solution	0.1	0.2	0.3	0.4	-
MI Chloroform	2.8	2.7	2.6	2.5	2.4
Diazo reagent	0.7	0.7	0.7	0.7	0.7
Methanol	3.5	3.5	3.5	3.5	3.5

RESULTS

RESULTS

1. AGE CRITERIA

AGE	MILD PRE-ECLAMPSIA		SEVERE PRE ECLAMPSIA		ECLAMPSIA		HELLP SYNDROME	
<20	4	5.8%	9	9%	4	12.5%	2	13.33%
20-24	22	32.35%	35	35%	13	40.63%	7	46.66%
25-29	32	47.06%	38	38%	10	31.25%	4	26.66%
30 & above	10	14.71%	18	18%	5	15.62%	2	13.33%

According to the above table there were 15 cases of HELLP Syndrome, most cases were in 20-24 years of age.

2. GRAVIDA

GRAVIDA	MILD PRE-ECLAMPSIA		SEVERE PRE ECLAMPSIA		ECLAMPSIA		HELLP SYNDROME	
Primi	40	58.82%	52	52%	17	53.12%	6	40%
Multi	28	41.18%	48	48%	15	46.88%	9	60%

According to the above table, HELLP Syndrome, shows higher incidence in multigravida

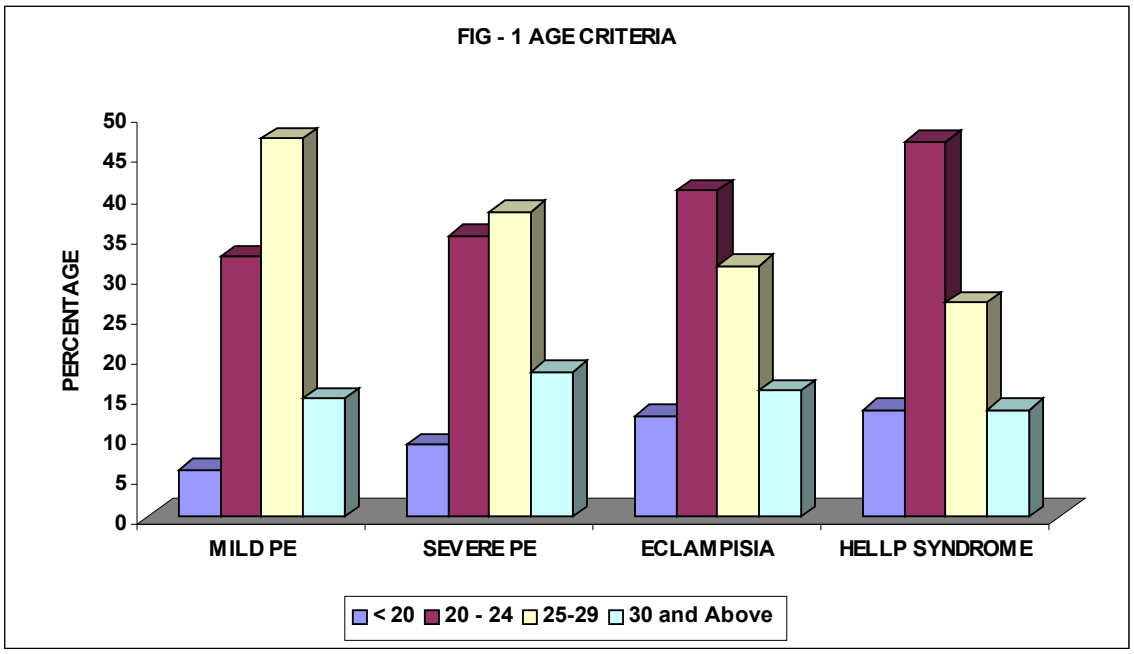


FIG - 1 AGE CRITERIA
PERCENTAGE
50
45
40
35
30
25
20
15
10
5
0
MILD PE
SEVERE PE
ECLAMPISIA
HELLP SYNDROME
< 20
20 - 24
25-29
30 and Above

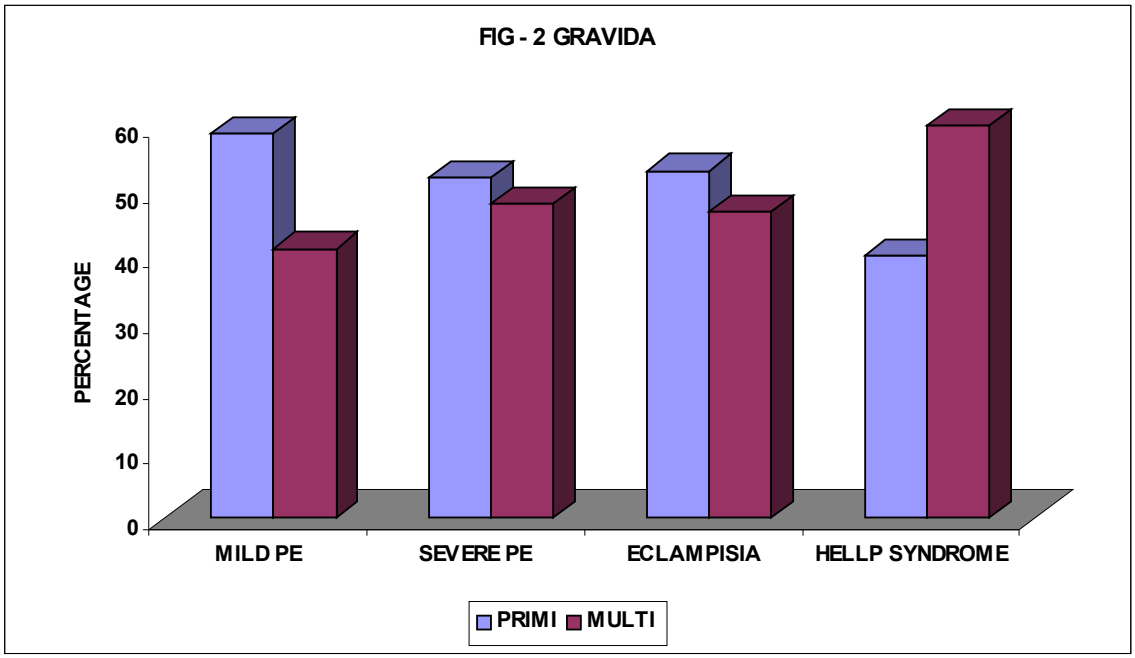


FIG - 2 GRAVIDA
PERCENTAGE
60
50
40
30
20
10
0
MILD PE
SEVERE PE
ECLAMPISIA
HELLP SYNDROME
PRIMI
MULTI

3. PROTEINURIA

PROTEINURIA	MILD PRE-ECLAMPSIA		SEVERE PRE ECLAMPSIA		ECLAMPSIA		HELLP SYNDROME	
Nil	-	-	-	-	-	-	-	-
Trace	48	70.59%	4	4%	2	6.25%	-	-
+	9	13.23%	36	36%	14	43.75%	1	6.66%
++	9	13.23%	30	30%	8	25%	7	46.66%
+++	2	2.94%	26	26%	6	18.75%	5	33.33%
++++	0	-	4	4%	2	6.25%	2	13.33%

There were significant proteinuria in 90% cases of HELLP Syndrome.

4. GESTATIONAL AGE

GESTATIONAL AGE	MILD PRE-ECLAMPSIA		SEVERE PRE ECLAMPSIA		ECLAMPSIA		HELLP SYNDROME	
<20	-	-	-	-	-	-	-	-
20-24	-	-	1	1%	-	-	-	-
25-29	3	4.41%	11	11%	4	12.5%	2	13.33%
30-24	15	22.06%	36	36%	12	37.5%	7	46.66%
35-39	50	73.53%	51	51%	16	50%	5	33.33%
Delivered	-	-	1	1%	-	-	1	6.66%

According to the above table HELLP Syndrome was more common in 30-34 weeks.

FIG - 3 PROTEINURIA

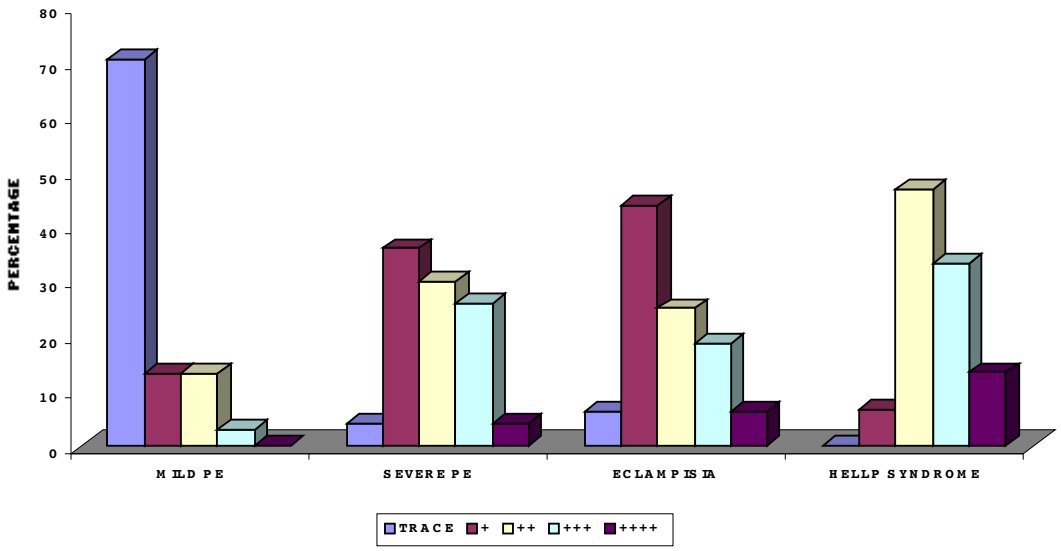
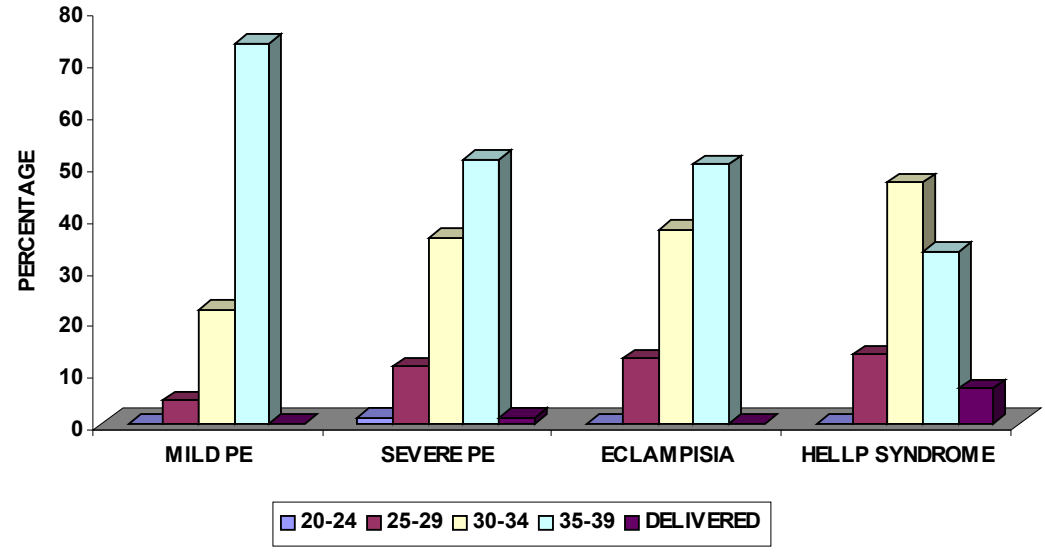


FIG - 4 GESTATIONAL AGE (WEEKS)



5. FUNDUS CHANGES

FUNDUS CHANGES	MILDPRE ECLAMPSIA		SEVERE PREECLAMPSIA		ECLAMPSIA		HELLP SYNDROME	
N ORMAL	67	98.53%	85	85%	27	84.37%	10	66.66%
GRADE I	1	1.47%	12	12%	3	9.38%	3	20%
GRADE II	-	-	3	3%	2	6.27%	2	13.33%

According to the above table, 67% cases of HELLP syndrome had normal fundus, 33% cases had fundus changes

6. PLATELETS

PLATELETS	MILDPRE ECLAMPSIA		SEVERE PREECLAMPSIA		ECLAMPSIA		HELLP SYNDROME	
CLASS I <50,000	-	-	-	-	-	-	-	-
CLASS II 50,000-1,00,000	-	-	2	2%	1	3.13%	3	20%
CLASS III 1,00,000-1,50,000	25	36.76%	55	55%	15	46.97%	12	80%
NORMAL >1,50,000	43	63.24%	43	43%	16	50%	-	-

Isolated thrombocytopenia were found in preeclamptic Eclamptic group but in HELLP syndrome 20% cases belonged to class II and 80% cases to class III

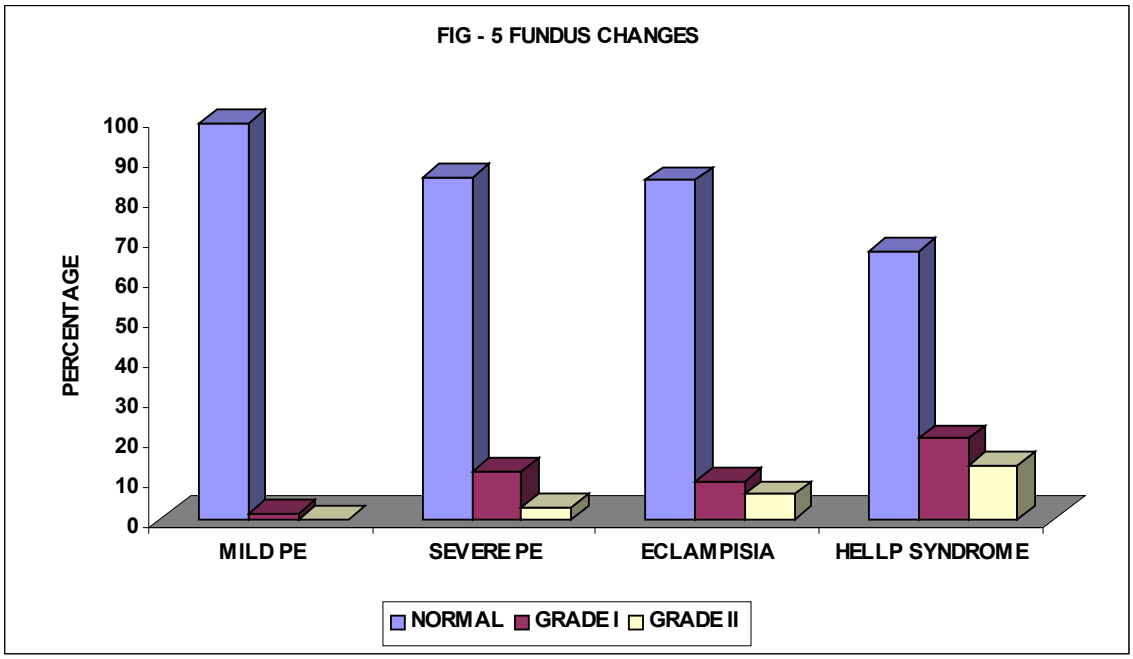


FIG - 5 FUNDUS CHANGES
PERCENTAGE
100
90
80
70
60
50
40
30
20
10
0
MILD PE
SEVERE PE
ECLAMPISIA
HELLP SYNDROME
NORMAL
GRADE I
GRADE II

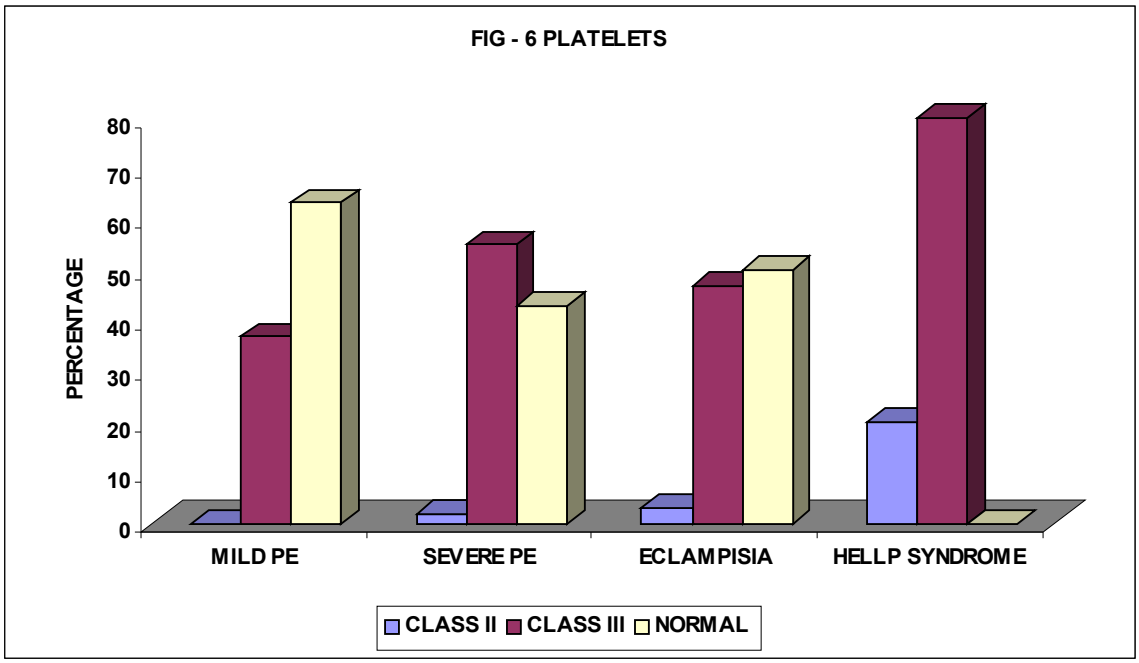


FIG - 6 PLATELETS
PERCENTAGE
80
70
60
50
40
30
20
10
0
MILD PE
SEVERE PE
ECLAMPISIA
HELLP SYNDROME
CLASS II
CLASS III
NORMAL

7. LIVER ENZYMES (ELEVATED)

LIVER ENZYMES (ELEVATED)	MILD PRE - ECLAMPSIA		SEVERE PRE - ECLAMPSIA		ECLAMPSIA		HELLP SYNDROME	
SGOT	5	7.35%	16	16%	8	25%	15	100%
SGPT	3	4.41%	16	16%	6	18.75%	15	100%
LDH	3	4.41%	13	13%	5	15.63%	15	100%

Isolated elevation of liver enzymes found in Preeclamptic - Eclamptic group but there was significant elevation of liver enzymes in HELLP syndrome.

8. MODE OF DELIVERY

MODE OF DELIVERY	MILD PRE - ECLAMPSIA		SEVERE PRE ECLAMPSIA		ECLAMPSIA		HELLP SYNDROME	
LN	38	55.88%	53	53%	9	28.12%	9	60%
FORCEPS	4	5.88%	2	2%	1	3.13%	1	6.66%
LSCS	20	29.4%	29	29%	18	56.25%	3	20%
EAC	6	8.82%	15	15%	4	12.5%	1	6.66%
CERVIPRIME	19	27.94%	26	26%	4	12.5%	3	20%
CAESAREAN HYSTEREC TOMY	-	-	1	1%	-	-	1	6.66%

According to the above table 60% cases of HELLP syndrome delivered by labour natural, 20% by LSCS, 6.7% by EAC (Preterm termination), 6.7% by caesarean hysterectomy.

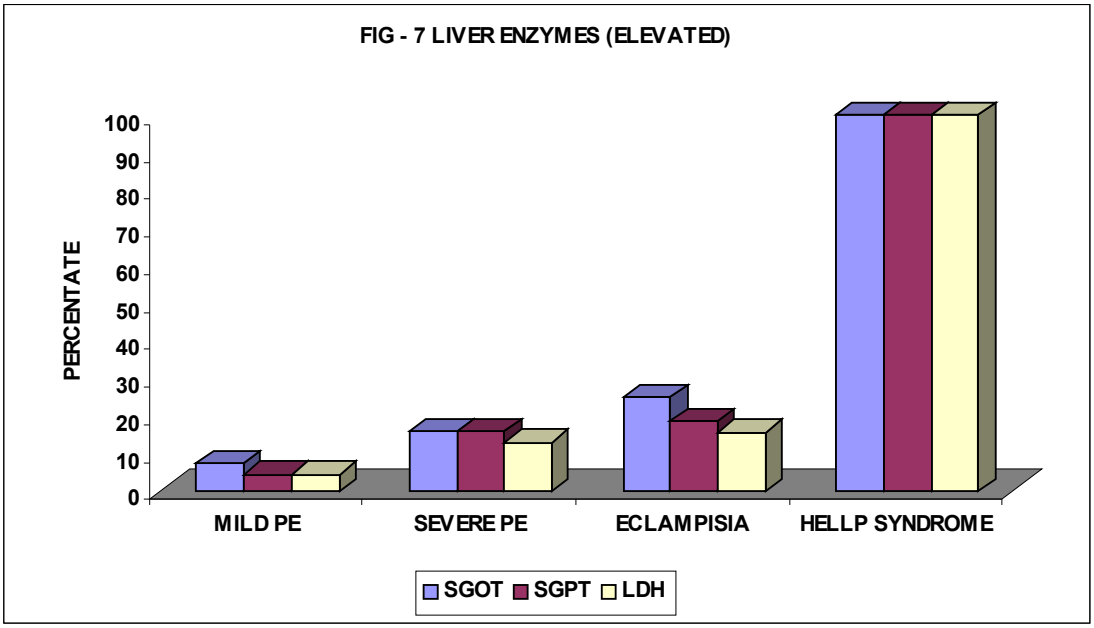


FIG - 7 LIVER ENZYMES (ELEVATED)
PERCENTATE
100
90
80
70
60
50
40
30
20
10
0
MILD PE
SEVERE PE
ECLAMPISIA
HELLP SYNDROME
SGOT
SGPT
LDH

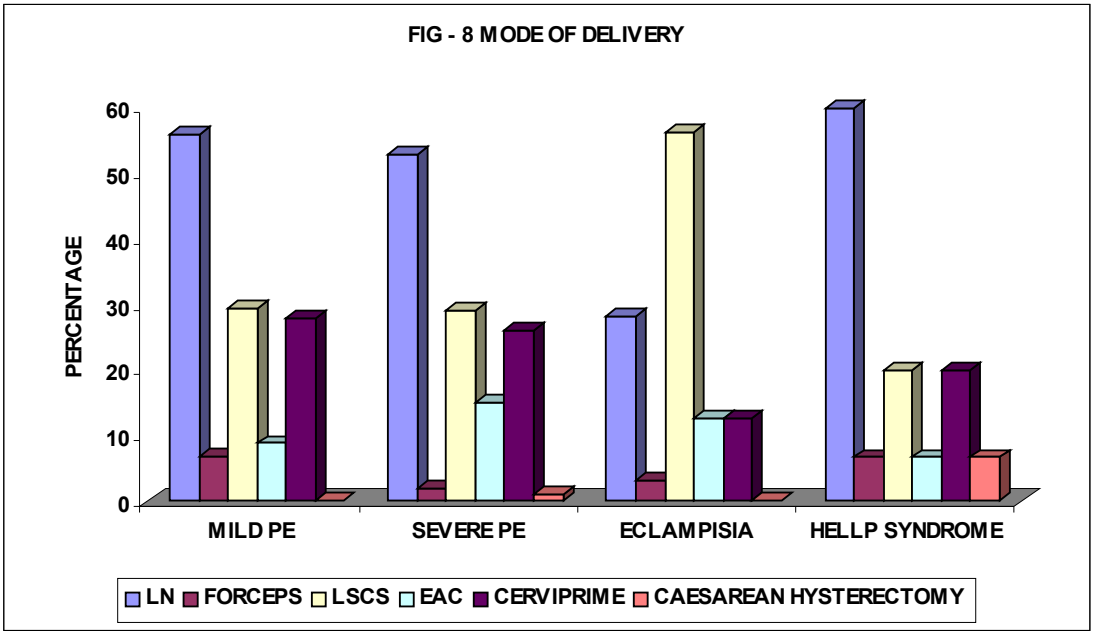


FIG - 8 MODE OF DELIVERY
PERCENTAGE
60
50
40
30
20
10
0
MILD PE
SEVERE PE
ECLAMPISIA
HELLP SYNDROME
LN
FORCEPS
LSCS
EAC
CERVIPRIME
CAESAREAN HYSTERECTOMY

9. MATERNAL OUTCOME

MATERNAL OUTCOME	MILD PRE ECLAMPSIA		SERVERE PRE ECLAMPSIA		ECLAMPSIA		HELLP SYNDROME	
ABRUPTION	3	4.41%	6	6%	2	6.25%	2	13.33%
CEREBRAL EDEMA	-	-	-	-	4	12.5%	1	6.66%
ACUTE RENAL FAILURE	1	1.47%	3	3%	-	-	2	13.33%
HIE	-	-	1	1%	1	3.12%	1	6.66%
CARDIORESPIRATORY ARREST	-	-	-	-	1	3.12%	1	6.66%
PERIPARTUM CARDIOMYOPATHY	-	-	1	1%	-	-	-	-
PULMONARY EDEMA	-	-	-	-	-	-	-	-

According to the above table, significant maternal morbidity in HELLP syndrome was due to abruption and acute renal failure.

10. PERINATAL OUTCOME

PERINATAL OUTCOME	MILD PRE – ECLAMPSIA		SEVERE PRE ECLAMPSIA		ECLAMPSIA		HELLP SYNDROME	
IUD	10	14.7%	20	20%	5	15.62%	5	33.33%
TERM ALIVE	38	55.88%	34 (1Twin)	34%	12	37.5%	4 (1Twin)	26.66%
TERM EXPIRED	-	-	2	2%	1	3.13%	-	-
PRETERM ALIVE	15	22.05%	34 (1Twin)	34%	9	28.13%	2	13.33%
PRETERM EXPIRED	3	4.41%	6	6%	5	15.62%	3	20%
IUGR ALIVE	2	2.94%	4	4%	-	-	2	13.33%
IUGR EXPIRED	-	-	2	2%	-	-	-	-

According to the above table, significant perinatal mortality in HELLP syndrome was due to prematurity

FIG - 9 MATERNAL OUTCOME

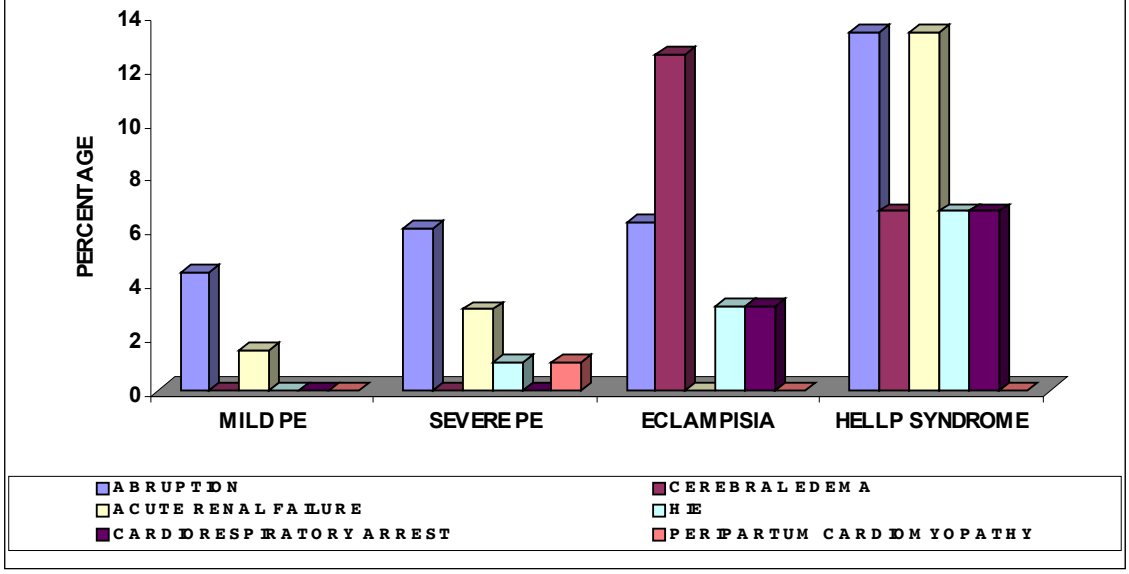
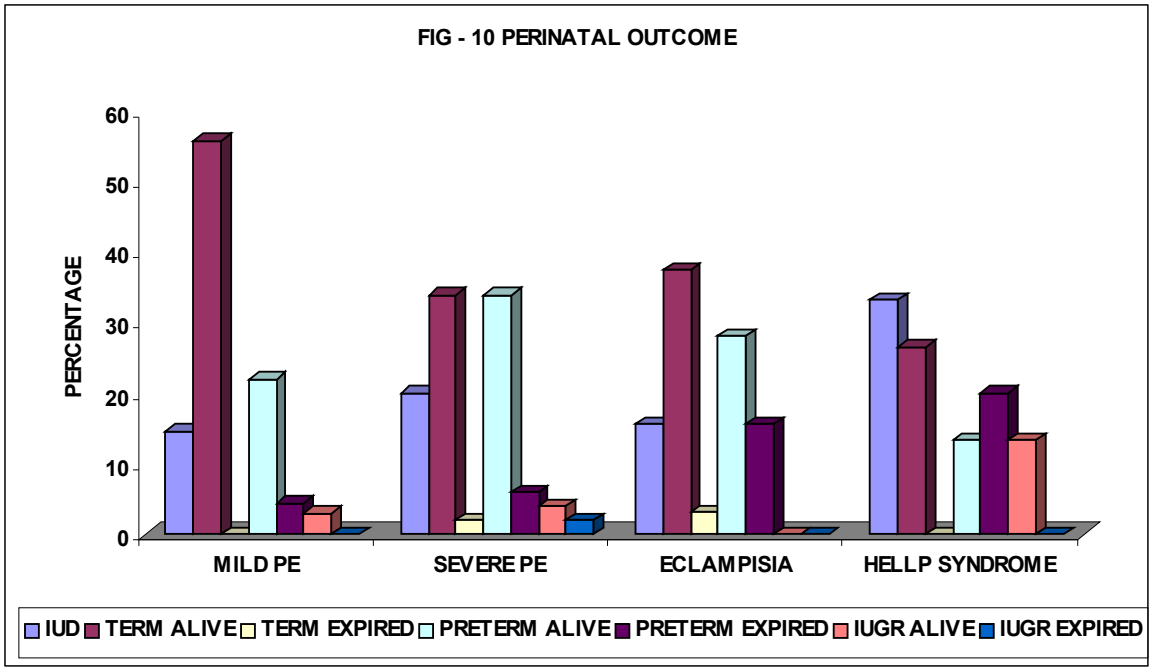


FIG - 10 PERINATAL OUTCOME



11. BIRTH WEIGHT

BIRTH WEIGHT (gms)	MILD PRE - ECLAMPSIA		SEVERE PRE – ECLAMPSIA		ECLAMPSIA		HELLP SYNDROME	
<500	-	-	-	-	-	-	-	-
500-999	2	2.94%	10	10%	2	6.25%	1	6.66%
1000-1499	10	14.70%	19	19%	4	12.5%	2	13.33%
1500-1999	11	16.18%	34	34%	8	25%	7	46.66%
2000 and above	45	66.18%	39	39%	18	56.25%	6	40%

Out of 16 babies delivered by HELLP syndrome cases, 6 babies were > 2000 gms

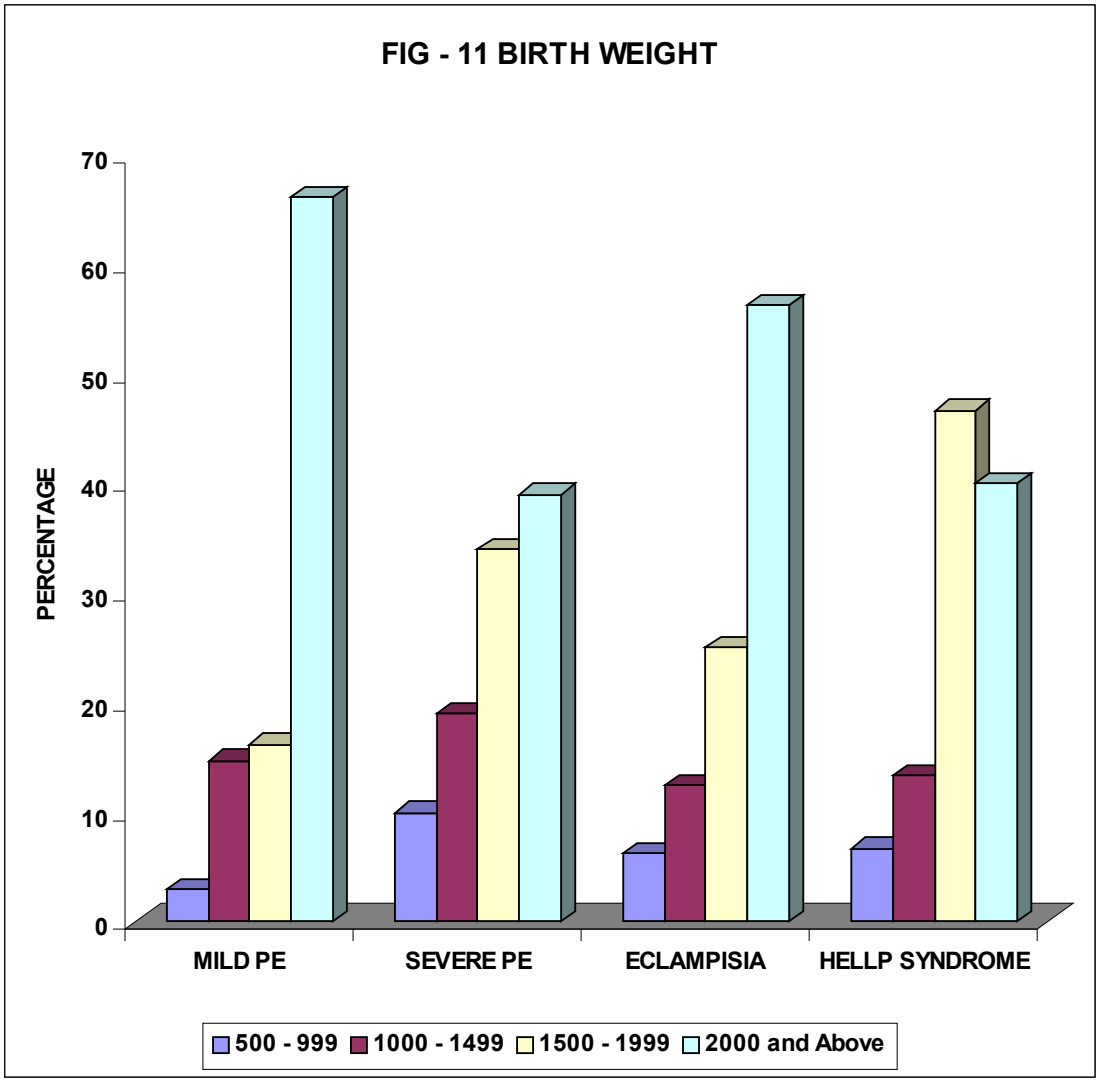


FIG - 11 BIRTH WEIGHT
PERCENTAGE
70
60
50
40
30
20
10
0
MILD PE
SEVERE PE
ECLAMPISIA
HELLP SYNDROME
500 - 999
1000 - 1499
1500 - 1999
2000 and Above

HELLP SYNDROME

A. BLOOD PRESSURE

MILD PREECLAMPSIA	SEVERE PREECLAMPSIA	ECLAMPSIA
1	11	3
6.66%	73.33%	20%

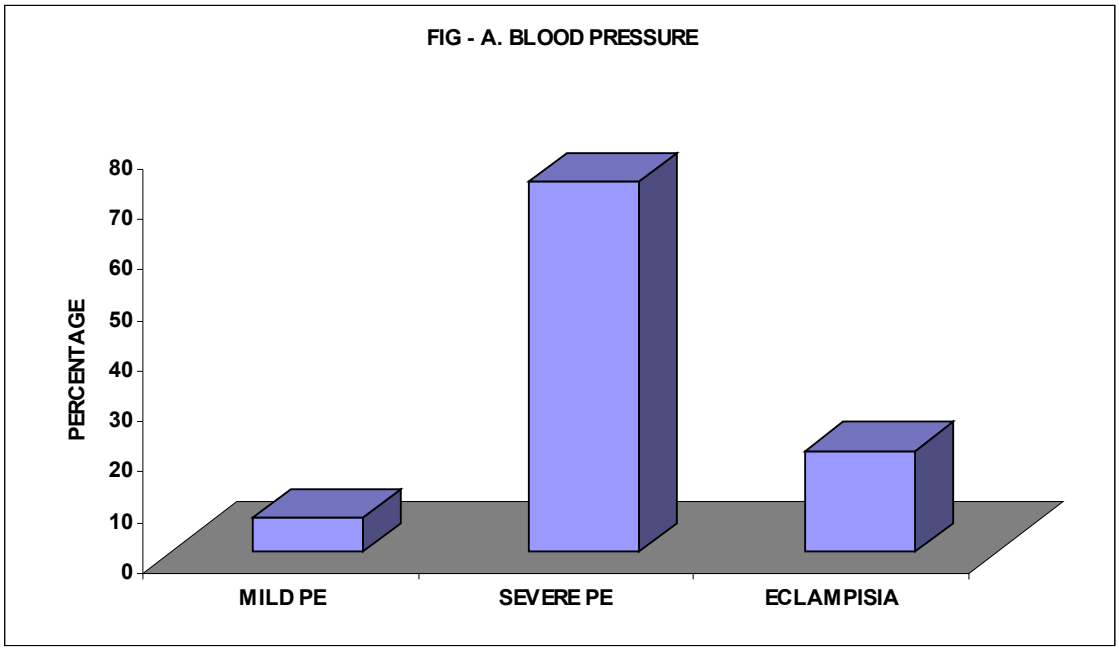
HELLP syndrome was common is severe preeclamptic group

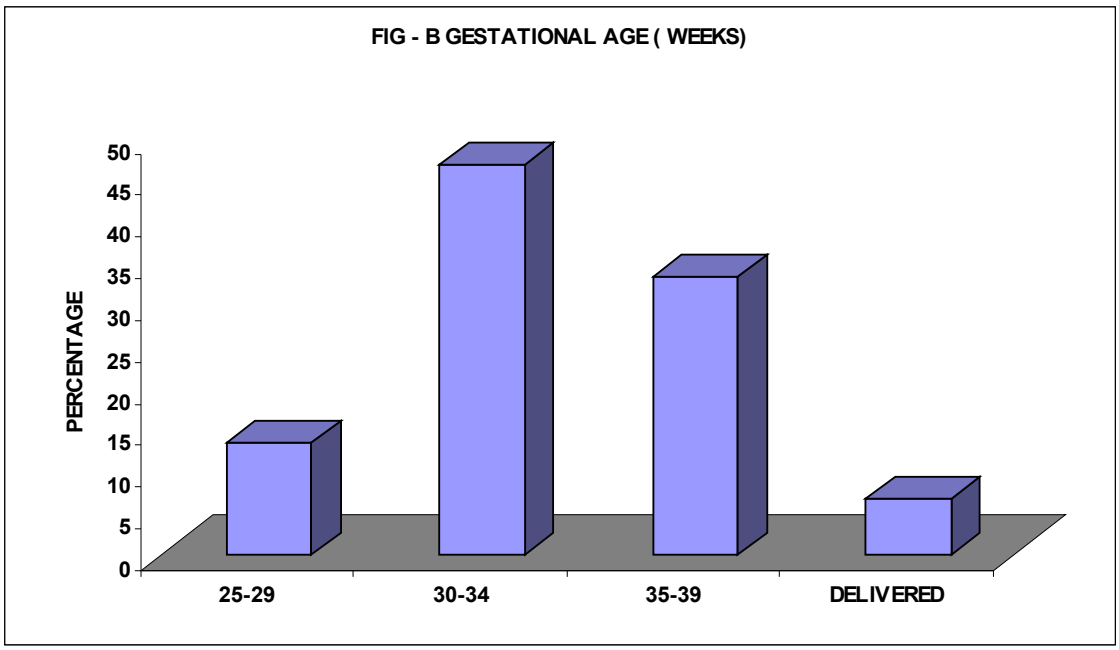
B. GESTATIONAL AGE

GESTATIONAL AGE (WEEKS)	HELLP SYNDROME	
< 20	-	-
20-24	-	-
25-29	2	13.33%
30-34	7	46.66%
35-39	5	33.33%
DELIVERED	1	6.66%

HELLP syndrome was common in 30-34 weeks gestation

HELLP SYNDROME





C. ANALYSIS OF LIVER ENZYMES LEVELS IN HELLP SYNDROME

Liver enzymes levels 1U/L	SGOT		SGPT	
70-110	8	53.33%	8	53.33%
111-150	3	20%	3	20%
151-190	1	6.66%	1	6.66%
191-230	-	-	-	-
231-270	2	13.33%	2	13.33%
271-310	-	-	-	-
311-350	-	-	-	-
351-390	1	6.66%	1	6.66%
391 & above	-	-	-	-

SGOT and SGPT levels in HELLP syndrome were found mostly in the range 70-110 IU/L

D. LDH

601-620 1U/L	621-640 1U/L	641-660 1U/L	661-680 IU/L	6810700 IU/L	Above 700 IU/L
3	4	2	3	3	-
20%	26.66%	13.33%	20%	20%	-

LDH levels mostly in the range 621 - 640 **1U/L**

FIG. - C ANALYSIS OF LIVER ENZYMES LEVELS IN HELLP SYNDROME

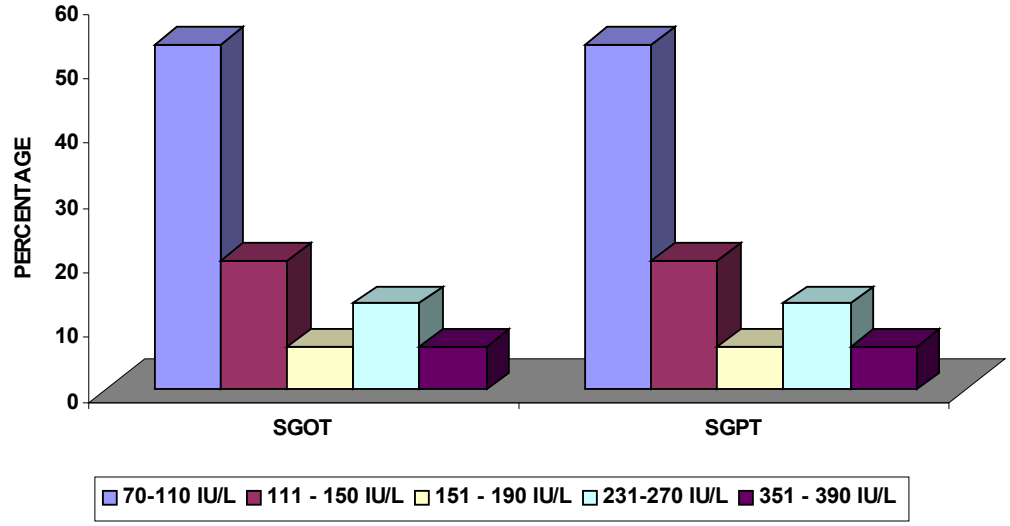
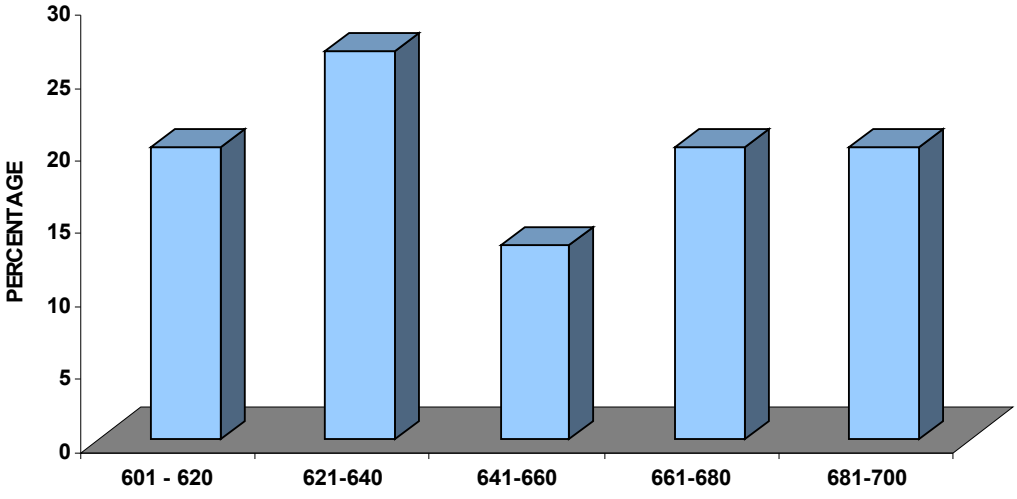


FIG. - D LDH (IU/L)

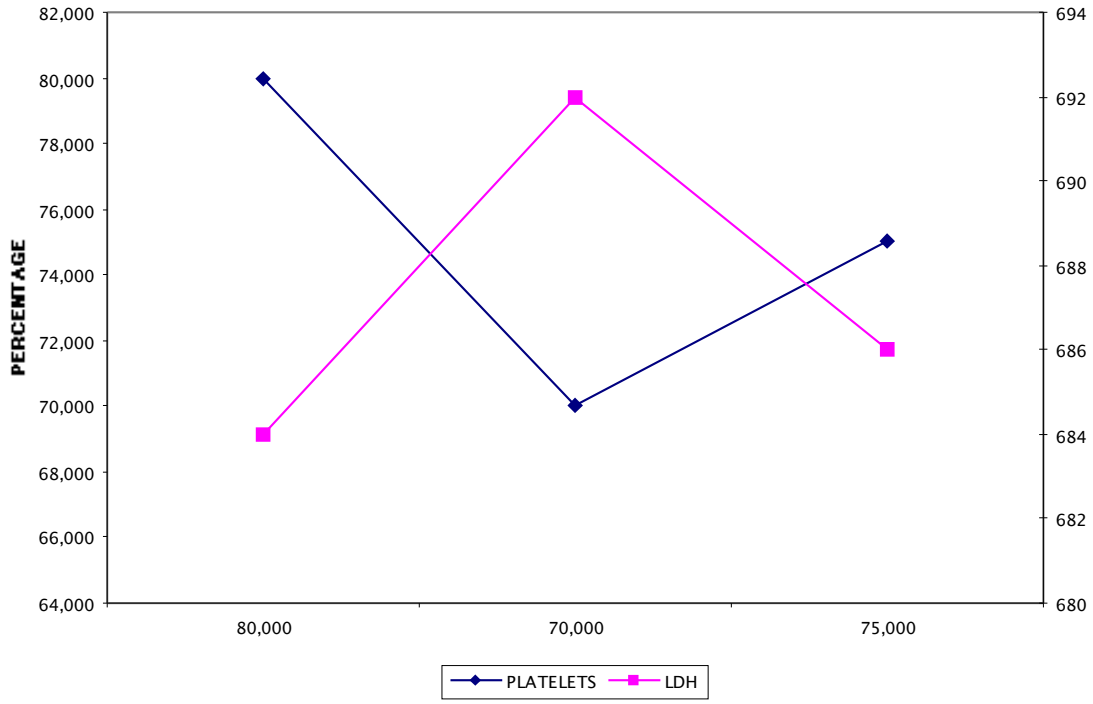


E. CORRELATION BETWEEN PLATELETS AND LIVER EZYMES IN CLASS II HELLP SYNDROME

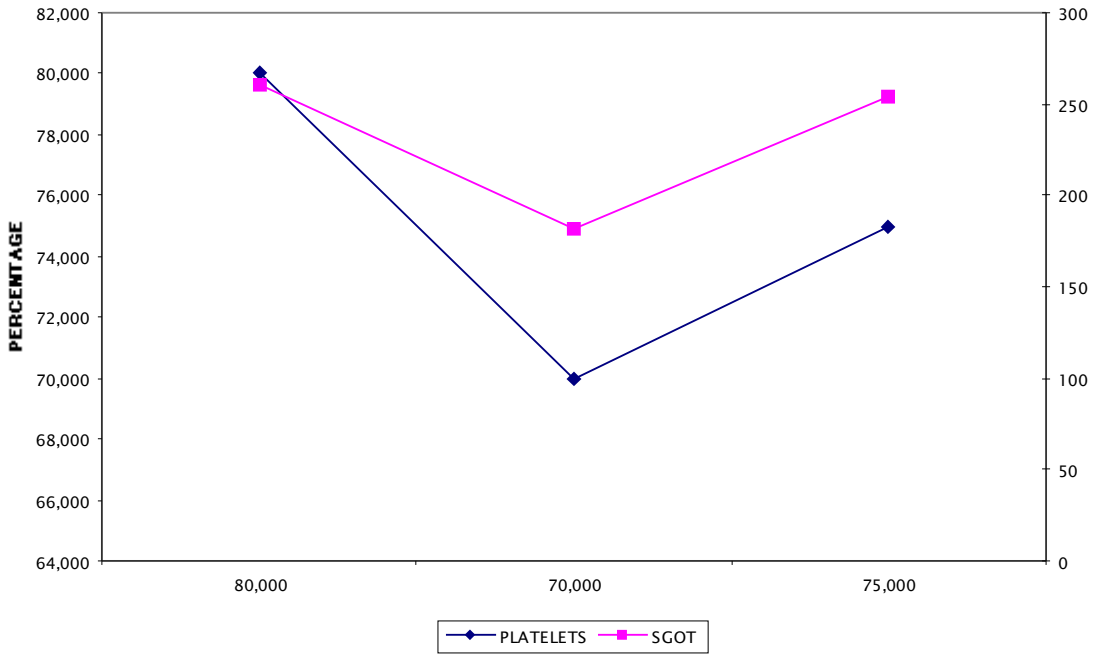
S.No.	PLATELETS	LDH IU/L	SGOT IU/L	SGPT IU/L
1.	80,000	684	261	265
2.	70,000	692	182	186
3.	75,000	686	254	256

There is inverse correlation between platelets and liver enzymes in class II HELLP syndrome.

Fig.E CORRELATION BETWEEN PLATELETS AND LDH IN CLASS II HELLP SYNDROME



CORRELATION BETWEEN PLATELETS AND SGOT IN CLASS II HELLP SYNDROME

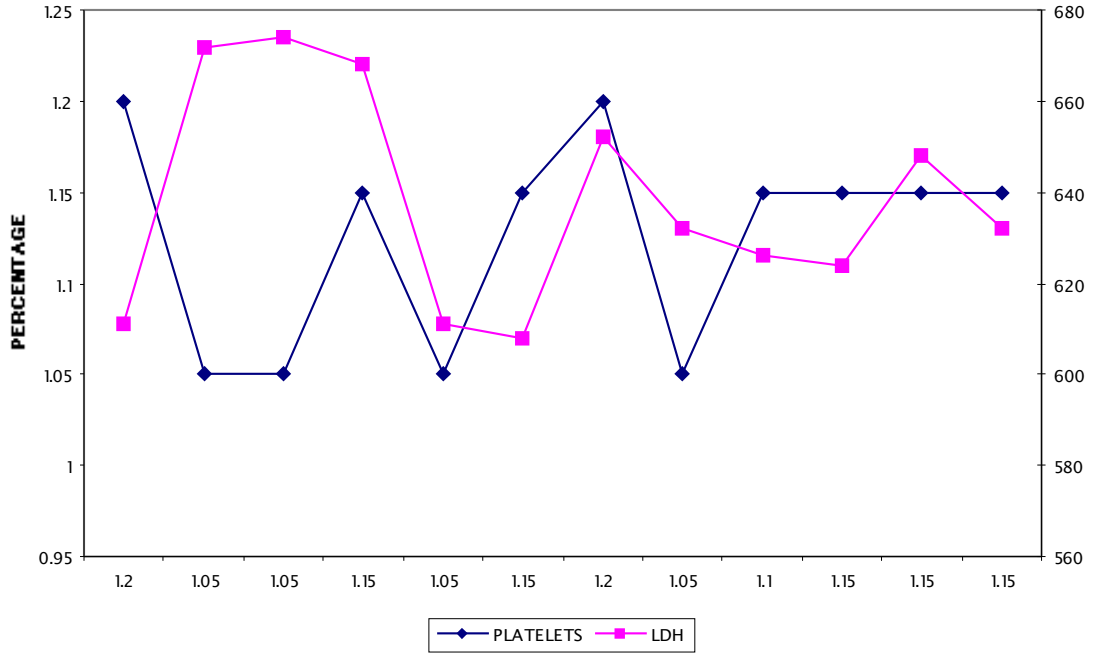


F. CORRELATION BETWEEN PLATELETS AND LIVER ENZYMES IN CLASS III HELLP SYNDROME

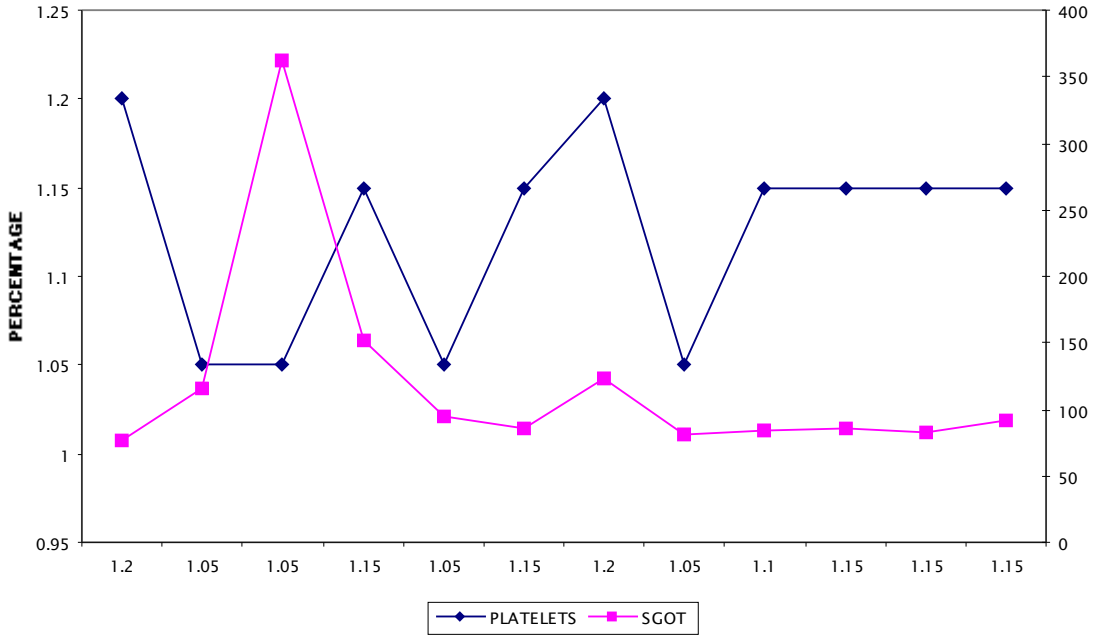
S.No.	Platelets	SGOT IU/L	SG PT IU/L	LDH 1U/L
1.	1.2	76	78	611
2.	1.05	116	118	672
3.	1.05	362	381	674
4.	1.15	152	148	668
5.	1.05	94	90	611
6.	1.15	86	88	608
7.	1.2	124	128	652
8.	1.05	81	84	632
9.	1.1	84	86	626
10.	1.15	86	84	624
11.	1.15	82	86	648
12.	1.15	92	96	632

There is inverse correlation between liver enzymes level and platelet count in class III HELLP syndrome.

FIG. F. CORRELATION BETWEEN PLATELETS AND LDH IN CLASS III HELLP SYNDROME



CORRELATION BETWEEN PLATELETS AND SGOT IN CLASS III HELLP SYNDROME



G. TREATMENT REGIMEN

ANTI HYPERTENSIVES + $MgSO_4$ REGIMEN	ANTIHYPERTENSIVES ONLY
10	5
66.66%	33.33%

H. ADMISSION - DELIVERY INTERVAL IN HELLP SYNDROME

< 6 hrs	6 - 10 hrs	11 -15 hrs	DELIVERED OUTSIDE
1	5	8	1
6.66%	33.33%	53.33%	6.66%

In all cases of HELLP syndrome, Admission Delivery interval was < 15 hrs

FIG - G TREATMENT REGIMEN

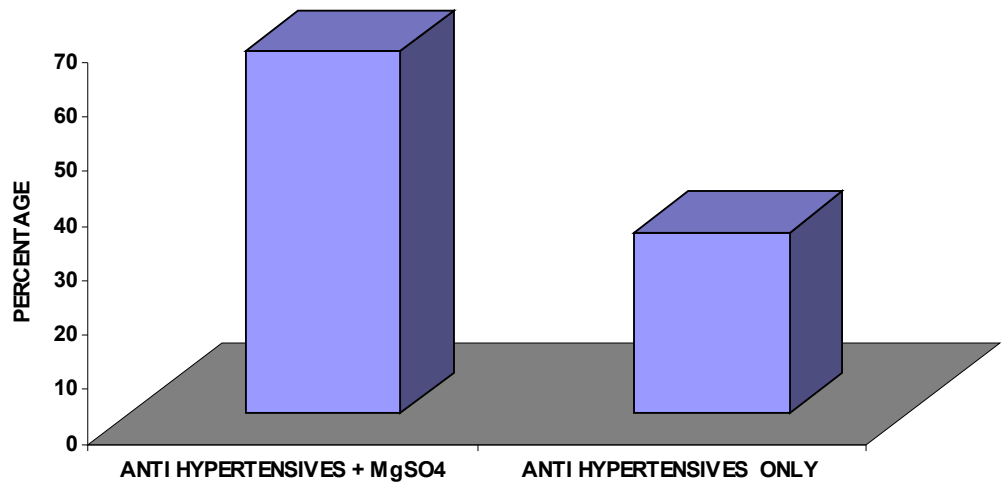
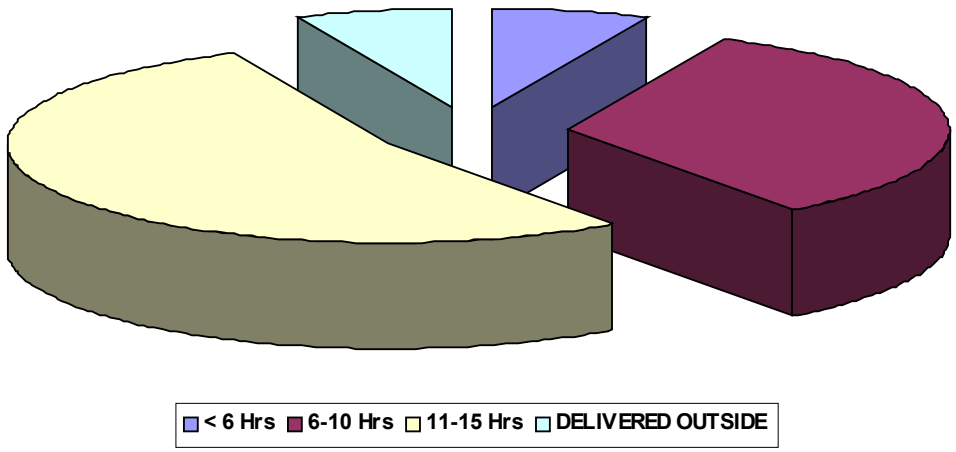


FIG - H ADMISSION - DELIVERY INTERVAL IN HELLP SYNDROME



I ANALYSIS OF MATERNAL MORTALITY

a. AGE

< 20	20-24	25-29	30 and above
-	2	1	-
-	66.66%	33.33%	-

b. GRAVIDA

All cases were primigravidae

c. BLOOD PRESSURE

MILD PREECLAMPSIA	SEVERE PRE ECLAMPSIA	ECLAMPISA
-	2	1
-	66.66%	33.33%

d. GESTATIONAL AGE

All cases were in 34 weeks gestation

e. PROTEINURIA

++	+++	++++
1	1	1
33.33%	33.33%	33.33%

f. PLATELETS COUNT

CLASS I < 50,000	CLASS II 50,000 - 1,00,000	CLASS III 1,00,000 - 1,50,000
-	2	1
-	66.6%	33.33%

g. LDH

All cases were in between 671-690 IU/L

h. SGPT & SGOT

231-270 (IU/L)	351-390 (IU/L)
2	1
66.66%	33.33%

i. MODE OF DELIVERY

FORCEPS	L.S.C.S	CAESAREAN HYSTERECTOMY
1	1	1
33.33%	33.33%	33.33%

j. ADMISSION-DELIVERY-DEATH INTERVAL

24 hrs	48 hrs
2	**1**
66.66%	**33.33%**

k. MATERNAL MORTALITY - CAUSES

ACUTE RENAL FAILURE	ABRUPTION, ACUTE RENAL FAILURE	ABRUPTION, CARDIO RESPIRATORY ARREST
1	1	1
33.33%	33.33%	33.33%

According to Table a-k, there were 3 cases of death in HELLP syndrome giving rise to maternal mortality rate of 20%. All cases were PRIMI between 20-25 years of age, mostly in severe preclampsia at 34 weeks gestation. All cases had significant proteinuria, delivered within 12 hrs of admission. Most common causes of death were abruption and acute renal failure.

POSTPARTUM LEVEL OF PLATELETS & LIVER ENZYMES IN HELLP SYNDROME

CASES	PLATELETS Lakhs . Cumm	SGOT IU/L	SGPT IU/L	LDH IU/L
CASE - 1				
Admission	1.2	76	78	611
PP Day 2	1.8	32	28	394
CASE – 2				
Admission	1.05	116	118	672
PP Day 3	1.75	22	28	391
CASE – 3				
Admission	1.15	152	148	668
PP Day 3	1.75	24	32	374
CASE – 4				
Admission	70,000	182	186	692
PP Day 4	1.6	26	30	380
CASE – 5				
Admission	1.05	94	90	611
PP Day 3	1.75	22	24	297
CASE 6				
Admission	1.15	86	88	608
PP Day 2	2.25	22	26	194

CASE 7				
Admission	1.2	124	128	652
PP Day 2	2.25	28	22	254
CASE – 8				
Admission	1.05	81	84	632
PP Day 3	1.95	24	26	248
CASE 9				
Admission	1.1	84	86	626
PP Day 3	1.95	28	24	236
CASE - 10				
Admission	1.15	86	84	624
PP Day 2	1.85	22	20	238
CASE -11				
Admission	1.15	82	86	648
PP Day 2	2.25	24	22	234
CASE - 12				
Admission	1.15	92	96	632
PP Day 3	2.25	22	26	248

In all cases of HELLP syndrome, platelets count and liver enzymes returned to normal limits within 4th postpartum day.

CONSOLIDATED ANALYSIS OF THE STUDY

STUDY		MILD PE		SEVERE PE		ECLAMPSIA		HELLP SYNDROME
		P	M	P	M	P	M	
Total No. of cases		40	28	52	48	17	15	15
Proteinuria		All	All	All	All	All	All	All
Fundus Changes		1	-	9	6	5	-	5
Low plateletcount		14	11	31	26	9	7	All
Evidence of haemolysis		-	1	5	6	1	2	15
Elevated liver enzymes		1	4	9	7	5	3	15
Mode of Delivery								
LN		22	16	25	28	3	6	9
Forceps		2	2	1	1	-	1	1
LSCS		13	7	17	12	13	5	3
EAC		3	3	8	7	1	3	1
Cerviprime		15	4	18	8	2	2	3
Caesarean Hysterectomy		-	-	1	-	-	-	1
Fetal outcome								
IUD		6	4	10	10	5	-	5
Term Alive		21	17	19	15	8	4	4
Term Expired		-	-	-	2	-	1	-
Preterm Alive		10	5	19	15	3	6	2
Preterm Expired		2	1	3	3	1	4	3
FGR alive		1	1	1	3	-	-	2
FGR Expired		-	-	1	1	-	-	-
Maternal outcome								
Abruption		1	2	4	2	2	-	2
ARF		1	-	3	-	-	-	2
HIE		-	-	1	-	-	1	1
Cerebral edema		-	-	-	-	1	3	1
Peripartum cardiomyopathy		-	-	1	-	-	-	-
Cardiorespiratory arrest		-	-	-	-	1	-	1
Pulmonary edema		-	-	-	-	-	-	-
HELLP SYNDROME	**Alive**	-	1	3	6	-	2	12
	Expired	-	-	2	-	1	-	3

CONSOLIDATED INFERENCE OF THE STUDY

Total no. of cases studied	:	200
Proteinuria	:	All (100%)
Fundus changes	:	21 (10.5%)
Low platelets count	:	98 (49%)
Elevated liver enzymes	:	29 (14.5%)
Evidence of haemolysis	:	15 (7.5%)

MODE OF DELIVERY

LN	:	100 (50%)
Forceps	:	7 (3.5%)
LSCS	:	67 (33.5%)
EAC	:	25 (12.5%)
Cerviprime	:	49 (24.5%)
Caesarean hysterectomy	:	1 (0.5%)

MATERNAL OUTCOME

Abruption	:	11 (5.5%)
Acute Renal failure	:	4 (2%)
HIE	:	2 (1%)
Cerebral edema	:	4 (2%)
Cardio respiratory arrest	:	1 (0.5%)
Peripartum Cardiomyopathy	:	1 (0.5%)
Pulmonary edema	:	Nil

PERINATAL OUTCOME

IUD	:	35 (17.5%)
Term Alive	:	84 (42%)
Term Expired	:	3 (1.5%)
Preterm Alive	:	58 (29%)
Preterm Expired	:	14 (7%)
IUGR Alive	:	6 (3%)
IUGR Expired	:	2 (1%)
Mild Preeclampsia	:	68 (34%)
Primi gravida	:	40
Multigravida	:	28
Severe Preeclampsia	:	100 (50%)
Primigravida	:	52
Multigravida	:	48
Eclampsia	:	32 (16%)
AP Eclampsia	:	24
Primi gravida	:	13
Multi gravida	:	11

IP Eclampsia	:	3
Primigravida	:	1
Multigravida	:	2
PP Eclampsia	:	5
Primi Gravida	:	3
Multi gravida	:	2

HELLP SYNDROME	**:**	**15 (7.5%)**
MATERNAL MORTALITY		
IN HELLP SYNDROME	**:**	**3 (20%)**
PERINATAL MORTALITY		
IN HELLP SYNDROME	**:**	**8 (53.33%)**

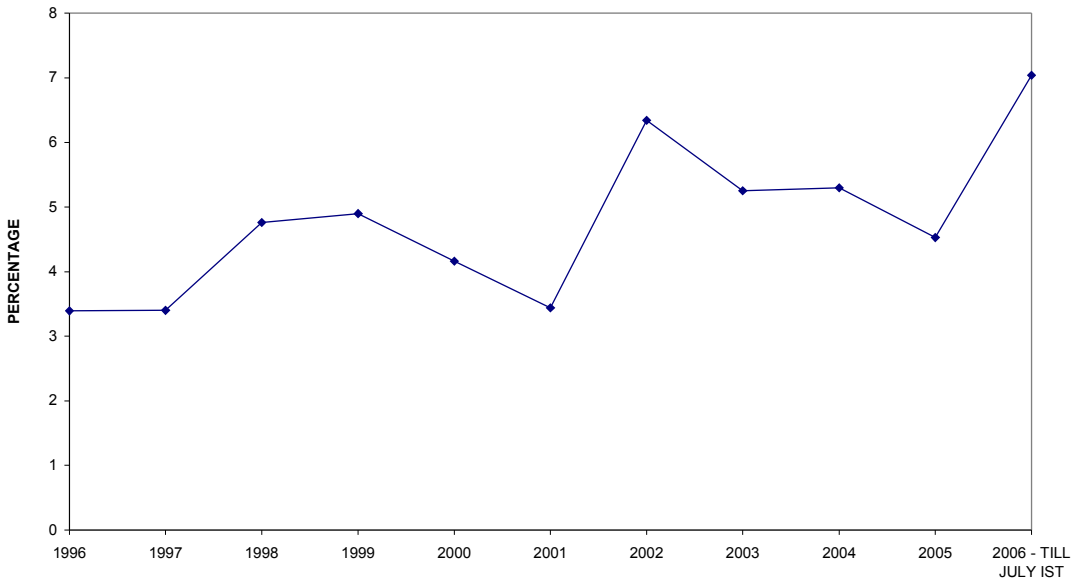
GOVERNMENT R.S. R.M. HOSPITAL

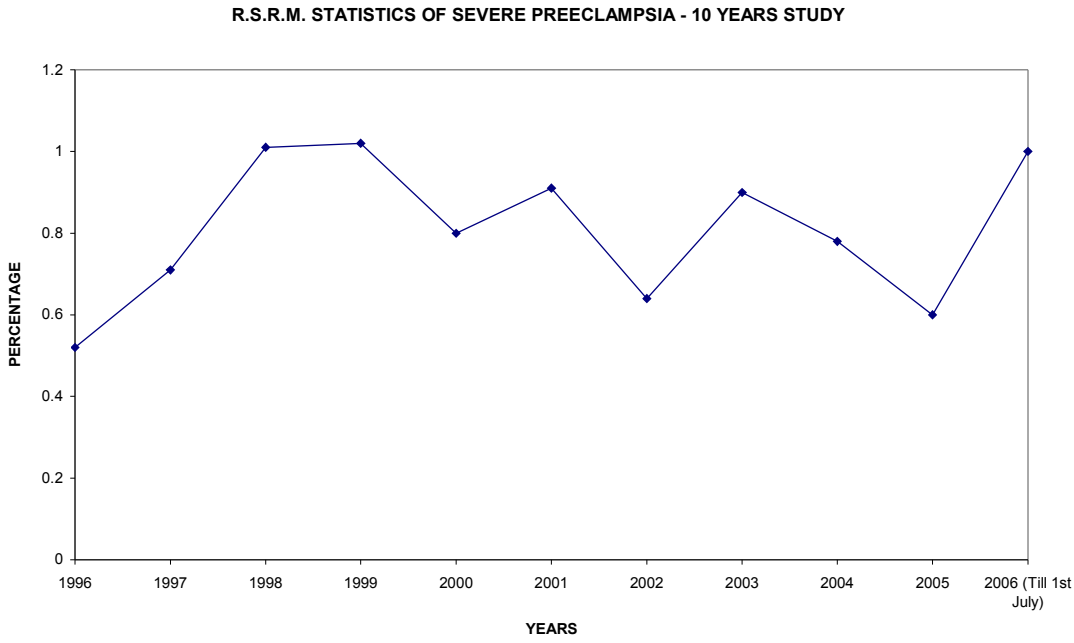
STATISTICS OF PRE ECLAMPSIA (MILD & SEVERE) AND ECLAMPSIA

- 10 YEARS STUDY

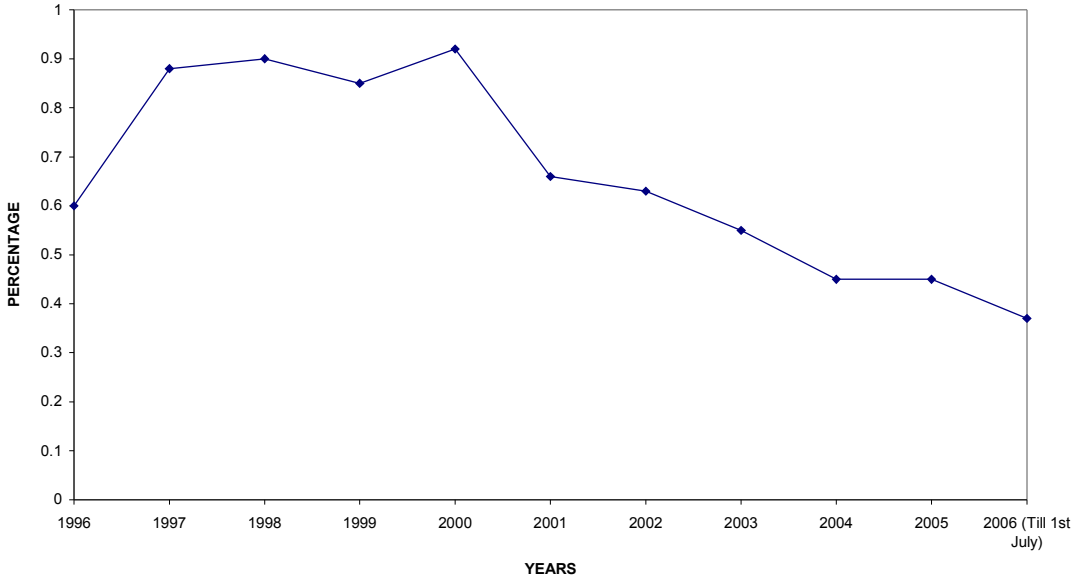
Year	Total No Deliveries	Mild Preeclampsia		Severe Preeclampsia		Eclampsia	
		No.	%	No.	%	No.	%
1996	10508	356	3.39	55	0.52	59	0.60
1997	10215	347	3.40	73	0.71	90	0.88
1998	10224	487	4.76	103	1.01	92	0.90
1999	10486	517	4.90	10.7	1.02	89	0.85
2000	12333	513	4.16	99	0.80	113	0.92
2001	12793	440	3.44	117	0.91	84	0.66
2002	12562	797	6.34	80	0.64	79	0.63
2003	14264	749	5.25	127	0.90	78	0.55
2004	14539	769	5.30	114	0.78	66	0.45
2005	14928	676	4.53	89	0.60	67	0.45
2006 till July 1st	7027	495	7.04	70	1.00	26	0.37

R.S.R.M. STATISTICS OF MILD PRE ECLAMPSIA - 10 YEARS STUDY




R.S.R.M. STATISTICS OF SEVERE PREECLAMPSIA - 10 YEARS STUDY
PERCENTAGE
1.2
1
0.8
0.6
0.4
0.2
0
1996
1997
1998
1999
2000
2001
2002
2003
2004
2005
2006 (Till 1st July)
YEARS

R.S.R.M. STATISTICS OF ECLAMPSIA - 10 YEARS STUDY



DISCUSSION

DISCUSSION

A study of 200 cases of preeclampsia -eclampsia was undertaken during the year 2005-2006 to identify all the patients with HELLP Syndrome. Of these 68 cases belonged to mild preeclampsia (34%), 100 cases belonged to severe preeclampsia (50%) and 32 cases belonged to eclampsia (16%).

Incidence

James N.Martin	4-12%
B.E. Reubnoff	12-13.6%
Sibai 1986	9.7%
Sibai & Mohammed 1993	18.9%
My study	**7.5%**

The wide range of incidence can be attributed to the remarkable variability of the diagnostic criteria of the syndrome. In addition, these incidence rates cannot be considered for general population since they are reported from tertiary referral centres

Age

University of Tennessee, Memphis	24.4 years
English literature	24.9 years
My study	**24.07 years**

Gravida

B.E. Reubinoff (1990)	Multipara
My study	**Multipara (60%)**

Incidence of HELLP Syndrome in multigravida (60%) is more than in primigravida (40%)

Socioeconomic status

All cases were unbooked and of low socioeconomic status probably because our institution caters of mainly to economically deprived strata of society.

Gestational age

The average, gestational age at presentation is 33.8 weeks, 70% of HELLP Syndrome patients manifest before labour, while 30% manifest after delivery. According

to Sibai 82% cases developed HELLP syndrome at <37 weeks. According to my study average gestational age at presentation is 33.86 weeks, of which 93.33% cases of HELLP syndrome manifest antepartum.

Clinical Symptoms & Signs

The frequent symptom is epigastric pain and /or right upper quadrant pain (90%) accompanied by nausea and vomiting. Headache may be present in 50% cases. In my study 50% cases had right upper quadrant pain associated with nausea and vomiting, rest of the patients (50%) had right upper quadrant pain and headache

Blood pressure

	Mild Pre eclampsia	**Severe pre eclampsia**	**Eclampsia**
Sibai et al	-	30%	10%
B.E. Reubinoff 1991	-	67%	-
My study	**6.66%**	**73.33%**	**20%**

According to my study, 6.66% cases of HELLP syndrome belonged to mild preeclampsia group, 73.33% to severe preeclampsia group and 20% belonged to eclampsia group. Surprisingly 1.47% of mild preeclamptic patients also developed HELLP Syndrome, indicating that HELLP Syndrome can also occur in mild preeclampsia patients (Dewhurst).

The difference between our results could be attributed to difference in the population studied (a large percentage of referred patients at one centre) and to differences in diagnostic criteria for HELLP Syndrome. The stressing factor here is, HELLP Syndrome should be anticipated even in the absence of hypertension and /or proteinuria.

Proteinuria

Urine analysis showed proteinuria of more than 2+ in 93.33% cases of HELLP Syndrome. It should be emphasized that 15% of HELLP Syndrome patients present with neither hypertension nor significant proteinuria (B.E. Reubinoff et al) In my study 6.66%

patients had 1+ poteinuria, 46.66% had 2+ proteinuria, 33.33% had 3 + proteinuria and 13.33% had 4+ proteinuria.

According to Sibai, some women with HELLP Syndrome however hypertension and proteinuria may be absent or slight. Thus it is imperative that all health care providers become knowledgeable about clinical signs & symptoms that might herald the onset of HELLP Syndrome.

Platelets levels in HELLP Syndrome

	Class I	**Class II**	**Class III**
Martin 1990	1.09%	28%	71%
My study	**-**	**20%**	**80%**

The definition of abnormal level varies among different studies. Severity of HELLP Syndrome is reflected in its laboratory parameters and not in the usual clinical parameters like blood pressure and proteinuria.

Haemolysis

Although microangiopathic hemolytic anemia underlies HELLP Syndrome, paradoxically most patients are not anemic when first admitted to the hospital. In my study all 15 cases had hemolytic blood picture with haemoglobin ranging between 7.8 gms - 10 gms.

Liver enzymes

According to B.E. Reubinoff et al., type of enzymes and definition of abnormal level also varies among different studies. There is direct correlation between the degree of thrombocytopenia and measures of liver dysfunction. An inverse correlation between platelets and LDH concentration was seen in both classes of HELLP Syndrome. Serum concentration of SGOT generally paralleled lactate dehydrogenase during the course of HELLP Syndrome. In my study there is inverse correlation between platelets and LDH in both classes of HELLP Syndrome.

Fundus Changes

In my study 5 cases of HELLP Syndrome had evidence of vasospasm and disc hyperemia at fundus examination (33.33%)

Treatment regimen

66.66% cases were started on $MgSO_4$ regimen in addition to antihypertensive, while 33.33% cases were in antihypertensive only.

Mode of delivery

According to Sibai (1999), caesarean delivery rate is high with HELLP Syndrome especially when pregnancy is less than 34 weeks of gestation (68%). In pregnancies less than 30 weeks of gestation caesarean section rate is 87%. According to my study, 60% cases of HELLP Syndrome delivered by Labour natural, 20% by LSCS 6.7% by EAC (Preterm termination), 6.7% by caesarean hysterectomy.

Admission Delivery interval

According to Martin (1990) Length of time between hospital admission and delivery with a mean of 15.6 hours. According to my study admission delivery interval was less than 15 hours in all cases of HELLP Syndrome.

Maternal Outcome

Though coagulapathy has been mentioned as the most common complication of HELLP Syndrome, in my study all parameters like bleeding time (B.T), Clotting time (C.T), serum fibrinogen were normal. This may be due to non sensitive parameters to detect DIC. More sensitive parameters like antithrombin IV, factor VIII and D-dimer may be needed to detect DIC.

	Abruption	Renal failure	Cerebral edema	Peri partum Cardiomyopathy
Sibai 1999	20%	8%	1%	6%
My study	**13.33%**	**13.33%**	**6.66%**	**-**

Maternal Mortality

According to sibai (1999) incidence of maternal mortality is as high as 24%. In my study, there were 3 cases of maternal death giving rise to maternal mortality rate of 20%, of these 1 case was due to cardio respiratory arrest and abruption 33.33%, 1 case was due to abruption and acute renal failure (33.33%) and 1 case was due to acute renal failure 33.33%.

Analysis of maternal mortality

Age-All cases were in 20-25 years of age.

Gravida - all cases were primigravida.

Blood pressure-66.66% cases belong to severe preeclampsia group, 33.33% cases belong to Eclampsia group.

Gestational age - All 3 cases were in 34 weeks gestation.

Proteinuria - All 3 patients had > 2+proteinuria.

Platelet count - 66.66% cases belong to class II HELLP Syndrome, 33.33% cases belong to class III HELLP Syndrome.

Liver enzymes - SGOT, SGPT and LDH were significantly elevated in all 3 cases.

Mode of delivery - 1 case (33.33%) delivered by outlet forceps with episiotomy, 1 case (33.33%) delivered by LSCS and 1 case (33.33%) underwent caesarean hysterectomy.

Admission - delivery death interval - 1 day for 2 cases (66.66%) and 2 days for 1 case (33.33%) In spite of early delivery there was 3 deaths in my study, probably due to late referral.

Perinatal outcome

According to Sibai perinatal mortality is 30-40% primarily because of prematurity. There is a significant trend for advanced form of HELLP Syndrome (Class I & Class II) to appear at earlier gestational age. According to my study perinatal mortality is 53.33%

primarily of prematurity.

Sibai 1999	30-40%
My study	**53.33%**

Birth weight of the baby

According to English literature, average weight of the new born is 1524-1898 gms and 30% were small for gestational age. According to my study 1 baby (6.66%) was 500-999 gms,2 babies (13.33) were 1000-1499 gms, 7 babies (46.66%) were 1500-1999 gms and 6 babies (40%) were 2000 grams and above.

Post partum period

Lowest platelet count did not predict peak values of aspartate aminotransferase or lactate dehydrogenase. The degree of abnormality of platelet counts, AST and LDH did not perspectively and accurately predict time of recovery. According to text, at 5-7 days after delivery, platelet count were above 1,00,000/cumm in 85-96% of cases, AST were below 70 IU/L in 85-96% and LDH values were below 600 IU/L in 76-89%. In my study all cases for platelet count and liver enzymes reverted to normal levels by 4th post partum day.

SUMMARY

SUMMARY

A prospective analytical study of 200 cases of preeclamptic -eclamptic group was undertaken during the year 2005-2006 to identify the patients at risk of HELLP Syndrome.

Incidence of HELLP Syndrome as per this study is 7.5%

Multigravida has a slightly higher incidence (60%) than primigravida (40%).

Mean maternal age of HELLP Syndrome as per this study is 24.07 years.

Average gestational age, that HELLP Syndrome presents is 33.86 weeks.

Most cases of HELLP Syndrome occurred antepartum (93.33%)

Most cases of HELLP Syndrome belonged to severe preeclamptic group (73.33%)

93.33% cases of HELLP Syndrome had > or = 2+ proteinuria.

20% cases of HELLP Syndrome belonged to class II and 80% cases belonged to

class III HELLP Syndrome.

Mode of delivery.

60% cases of HELLP Syndrome delivered by labour natural, 20% by LSCS, 6.7% by caesarean hysterectomy 6.7% cases were terminated by extra amniotic catheter before term.

Admission delivery interval was <15 hours in all cases.

Significant maternal morbidity in HELLP Syndrome was due to abruption(13.33%) and acute renal failure (13.33%).

Significant pertinatal morbidity and mortality in HELLP Syndrome was due to prematurity.

Maternal mortality rate in HELLP Syndrome is 20% Perinatal Mortality rate in HELLP Syndrome is 53.33%

In all cases, the laboratory parameters returned to normal limits within 4th postpartum day.

CONCLUSION

CONCLUSION

The question of whether HELLP Syndrome exists as a distrint entity or not is a part of a spectrum of pregnancy complications, which have common liver dysfunction, haemolysis and thrombocytopenia has long been a source of speculation among obstetricians and physicians. However, the importance of this collection of signs and symptoms lies not in its name but rather in its associated high maternal and perinatal morbidity and mortality. Hence,

1. Patient with HELLP Syndrome warrants an emergency obstetric help.

2. HELLP Syndrome demands, careful and close evaluation of maternal and neonatal parameters and should be given equal attention in decision making.

3. Prompt delivery is mandatory regardless of gestational age.

4. Successful management requires recognition, a timely intervention and to render optimal patient treatment.

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PROFORMA

PROFORMA

1. Name of the patient :
2. Age :
3. DOA :
4. D.O.D. :
5. Married :
6. Socio Economic Group :
7. Gravida :
8. Para :
9. Abortion :
10. L.M.P. :
11. EDD :
12. H/o. M.A. :
13. F/H HT :
    Cardiac disease :
    D.M :
14. Present illness :
    a. Giddiness :
    b. Oliguria :
    c. Blurring in Vision :

d. Head Ache :

e. Epi gastric pain :

15. G/E

a. Anaemia :

b. Oedema :

c. Pulse :

d. BP :

16. O/E

a. Ascites :

b. Any overdistension :

c. Height of uterus :

d. Head engaged (or) not :

e. F.H. :

f. Amount of liquor :

g. Abnormal Presentation :

h. Complication Abruption :

17. Investigations

a. Daily weight :

b. Urine C & S :

c. Albumin deposit :

d Fundus examination :

e. Blood Urea :

f. Sr.Uric Acid :

g. Sr.Creatinine :

h. Peripheral Smear :

i. anisocytosis :

ii. Poikilocytosis :

iii. Schistocytosis :

iv.Burr Cells :

v.Micro Spherocyte :

18. Serum Bilirubin :

Total Bilirubin :

Direct Bilirubin :

19. SGOT :

SGPT :

Sr. LDH :

20. Platelet count :

21. USG :

22. Outcome of delivery :

Mode of delivery :

a. Natural delivery : :

b. Forceps :

c. L.S.C.S. :

22. Baby Wt, Apgar

MASTER CHART

MILD PREECLAMPSIA - PRIMI

Sl. No.	Age	Gestational age (weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg %	Sr. Creatinine mg %	Serum Uric acid mg %	Platelet Count Lakhs / mm³	Peripheral Smear	Sr. Bilirubin mg %			SG OT IU/L	SG PT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
												Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
1.	19	36	49	+	140/100	Normal	16	0.6	4.1	1.75	NHBP	0.9	0.6	0.3	22	24	102	-	-	✓c	M	2.7	8/10	Well	Well
2.	22	34	52	Trace	150/90	Normal	18	0.6	3.5	1.05	NHBP	0.8	0.5	0.3	30	32	116	✓	-	-	F	1.9	7/10	Well	Well
3.	20	35	50	Trace	140/90	Normal	17	0.6	3.5	1.85	NHBP	0.8	0.6	0.2	30	28	122	✓c	-	-	F	1.85	7/10	Well	Well
4.	23	37	62	+	140/100	Normal	20	0.7	3.6	1.15	NHBP	0.9	0.7	0.2	26	24	147	-	-	✓c	M	2.8	8/10	Well	Well
5.	32	32	56	Trace	130/100	Normal	18	0.6	3.7	2.25	NHBP	0.9	0.6	0.3	28	30	98	EAC	-	-	F	1.4	8/10	Well	Well
6.	21	37	48	++	150/100	Normal	16	0.6	3.6	2.15	NHBP	0.9	0.6	0.3	24	28	91	-	-	✓	M	2.6	8/10	Abruptio Well	Well
7.	26	38	64	Trace	140/100	Normal	22	0.6	3.5	1.25	NHBP	0.8	0.6	0.2	30	34	106	-	-	✓c	M	3.5	8/10	Well	Well
8.	25	34	56	Trace	130/100	Normal	20	0.6	3.3	2.05	NHBP	0.8	0.5	0.3	28	32	104	✓	-	-	F	1.8	8/10	Well	Well
9.	27	40	63	+	140/90	Normal	24	0.7	3.3	1.05	NHBP	0.9	0.6	0.3	20	22	96	✓c	-	-	M	3.0	8/10	Well	Well
10.	17	28	47	Trace	150/90	Normal	18	0.6	3.5	2.2	NHBP	0.9	0.7	0.2	73	75	607	EAC	-	-	F	950 gm	1/10	Well	Expired
11.	25	40	61	++	150/100	Grade I	16	0.6	3.7	1.25	NHBP	0.8	0.6	0.2	22	24	126	-	-	✓	M	3.75	8/10	Well	Well
12.	24	38	49	Trace	140/100	Normal	18	0.6	3.3	2.4	NHBP	0.9	0.6	0.3	26	22	137	✓c	-	-	F	3.2	7/10	Well	Well
13.	31	30	61	Trace	140/100	Normal	20	0.6	4.1	1.45	NHBP	0.8	0.6	0.2	28	24	143	✓	-	-	M	1.2	2/10	Well	Expired

Sl. No.	Age	Gestational age (weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg %	Sr. Creatinine mg %	Serum Uric acid mg %	Platelet Count Lakhs / mm³	Peripheral Smear	Sr. Bilirubin mg %			SG OT IU/L	SG PT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
												Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
14.	27	36	60	+	150/100	Normal	21	0.7	4.0	2.65	NHBP	0.8	0.5	0.3	22	20	127	✓	-	-	F	2.3	7/10	Well	Well
15.	23	35	57	Trace	150/90	Normal	19	0.6	3.5	1.95	NHBP	0.9	0.7	0.2	22	24	118	✓	-	-	F	1.75	IUD	Well	Dead Born
16.	28	37	59	++	130/100	Normal	20	0.6	3.7	1.35	NHBP	0.8	0.5	0.3	30	26	116	✓	-	-	F	1.9	7/10	Well	Well
17.	22	34	56	Trace	140/100	Normal	22	0.6	3.3	1.75	NHBP	0.8	0.5	0.3	32	28	124	✓	-	-	F	1.85	7/10	Well	Well
18.	27	37	54	Trace	140/100	Normal	20	0.6	3.5	2.25	NHBP	0.9	0.6	0.3	30	34	134	-	✓c	-	M	2.75	8/10	Well	Well
19.	18	38	51	+	150/100	Normal	18	0.6	3.7	2.15	NHBP	0.9	0.7	0.2	32	34	147	✓	-	-	M	2.2	IUD	Well	Dead Born
20.	20	37	49	+	140/90	Normal	18	0.6	3.3	1.95	NHBP	0.8	0.6	0.2	26	22	106	-	-	✓	M	2.6	8/10	Well	Well
21.	29	38	56	Trace	150/90	Normal	20	0.6	3.3	1.25	NHBP	0.9	0.6	0.3	24	26	104	-	✓	-	M	2.75	7/10	Well	Well
22.	27	32	56	Trace	150/100	Normal	20	0.6	3.5	1.85	NHBP	0.8	0.5	0.3	28	22	114	EAC	-	-	F	1.45	IUD	Well	Dead Born
23.	32	38	64	++	140/100	Normal	18	0.6	3.1	2.25	NHBP	0.8	0.5	0.3	24	20	116	✓C	-	-	F	3.1	8/10	Well	Well
24.	25	32	57	Trace	130/100	Normal	22	0.7	3.7	1.35	NHBP	0.9	0.7	0.2	20	20	117	✓	-	-	F	1.4	7/10	Well	Well
25.	21	37	61	Trace	130/100	Normal	24	0.6	3.5	2.35	NHBP	0.9	0.6	0.3	22	26	126	✓	-	-	M	2.3	IUD	Well	Dead Born
26.	26	40	58	Trace	140/90	Normal	22	0.7	3.3	2.45	NHBP	0.9	0.6	0.3	30	28	114	-	-	✓	M	3.8	8/10	Well	Well

Sl. No.	Age	Gestational age (weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg %	Sr. Creatinine mg %	Serum Uric acid mg %	Platelet Count Lakhs / mm³	Peripheral Smear	Sr. Bilirubin mg %			SG OT IU/L	SG PT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
												Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
27.	22	30	53	Trace	150/90	Normal	18	0.6	4.1	1.15	NHBP	0.8	0.6	0.2	32	30	96	✓	-	-	F	1.3	7/10	Well	Well
28.	27	37	59	++	140/100	Normal	20	0.6	4.0	2.25	NHBP	0.8	0.6	0.2	30	24	98	-	-	✓C	M	2.7	8/10	Well	Well
29.	26	38	61	Trace	140/90	Normal	22	0.6	3.7	2.15	NHBP	0.8	0.5	0.3	32	22	134	✓C	-	-	M	2.5	8/10	Well	Well
30.	18	36	47	++	150/100	Normal	24	0.6	3.5	1.05	NHBP	0.9	0.6	0.3	34	30	146	✓	-	-	M	2.1	IUD	ARF Well	Dead Born
31.	25	34	53	Trace	150/90	Normal	18	0.6	3.7	2.05	NHBP	0.9	0.7	0.2	18	20	154	✓	-	-	F	1.75	8/10	Well	Well
32.	22	36	54	Trace	150/100	Normal	16	0.6	3.3	1.2	NHBP	0.9	0.7	0.2	20	22	152	-	-	✓C	F	2.4	8/10	Well	Well
33.	26	35	59	Trace	150/90	Normal	19	0.6	3.5	2.15	NHBP	0.9	0.6	0.3	24	20	150	✓	-	-	F	2.0	8/10	Well	Well
34.	23	36	60	+	140/90	Normal	17	0.6	4.0	2.35	NHBP	0.9	0.6	0.3	26	22	147	-	-	✓	M	2.3	8/10	Well	Well
35.	28	34	61	Trace	140/100	Normal	16	0.6	4.1	2.45	NHBP	0.8	0.6	0.2	28	26	143	✓	-	-	F	1.8	8/10	Well	Well
36.	27	37	63	Trace	150/90	Normal	20	0.6	4.2	1.35	NHBP	0.8	0.5	0.3	30	24	137	✓C	-	-	F	2.5	7/10	Well	Well
37.	22	38	57	++	140/100	Normal	18	0.6	3.7	2.5	NHBP	0.8	0.5	0.3	24	22	135	-	-	✓	M	2.7	7/10	Well	Well
38.	26	38	64	Trace	130/100	Normal	22	0.7	3.5	1.95	NHBP	0.8	0.6	0.2	28	26	126	✓C	-	-	M	2.65	8/10	Well	Well
39.	21	38	54	Trace	130/100	Normal	22	0.6	4.5	2.15	NHBP	0.9	0.6	0.3	22	24	126	-	-	✓c	F	2.5	8/10	Well	Well
40.	30	37	57	+	150/100	Normal	24	0.6	4.1	1.35	NHBP	0.8	0.6	0.2	28	26	114	-	-	✓c	M	2.3	IUD	Well	Dead Born

MILD PREECLAMPSIA - MULTI

Sl. No.	Age	Gestational age (Weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg %	Sr. Creatinine mg%	Serum Uric acid mg%	Platelet Count Lakhs / mm³	Peripheral Smear	Sr. Bilirubin mg %			SG OT IU/L	SG PT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
												Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
1.	24	34	54	Trace	140/100	Normal	18	0.6	3.3	1.15	NHBP	0.9	0.6	0.3	26	28	191	✓	-	-	F	1.95	7/10	Well	Well
2.	26	37	59	Trace	140/100	Normal	19	0.6	3.1	1.65	NHBP	0.8	0.6	0.2	22	24	101	✓	-	-	M	2.6	8/10	Well	Well
3.	30	38	67	++	140/100	Normal	18	0.7	3.5	1.35	NHBP	0.9	0.7	0.2	24	20	171	✓	-	-	M	2.6	8/10	Well	Well
4.	23	30	60	Trace	140/100	Normal	20	0.6	3.7	1.85	NHBP	0.8	0.5	0.3	20	22	98	✓	-	-	F	1.45	7/10	Well	Well
5.	25	36	62	Trace	130/90	Normal	16	0.6	3.5	1.25	NHBP	0.8	0.5	0.3	19	24	106	✓	-	-	M	2.2	8/10	Well	Well
6.	27	35	59	Trace	140/90	Normal	18	0.6	4.0	1.95	NHBP	0.9	0.6	0.3	77	81	603	EAC	-	-	M	2.05	IUD	Abruption Well	Dead born
7.	31	37	63	Trace	150/100	Normal	20	0.6	4.2	2.25	NHBP	0.8	0.6	0.2	24	22	167	-	-	✓	F	2.75	8/10	Well	Well
8.	22	32	58	+++	140/100	Normal	22	0.7	3.7	1.05	NHBP	0.8	0.5	0.3	26	22	154	✓	-	-	M	1.4	7/10	Well	Well
9.	26	38	54	Trace	150/90	Normal	20	0.6	3.5	2.15	NHBP	0.8	0.6	0.2	24	20	176	✓C	-	-	F	2.9	8/10	Well	Well
10.	28	40	62	Trace	150/100	Normal	24	0.7	3.5	2.05	NHBP	0.9	0.6	0.3	22	22	164	-	-	✓c	M	3.2	9/10	Well	Well
11.	31	38	49	Trace	140/90	Normal	18	0.6	3.3	2.15	NHBP	0.9	0.6	0.3	24	20	91	✓	-	-	M	2.8	8/10	Well	Well
12.	23	39	56	+	140/90	Normal	19	0.6	6.8	1.2	Schistocytes+	0.8	0.5	0.3	76	78	611	-	-	✓	F	3.1	8/10	Well	Well
13.	24	37	47	Trace	140/100	Normal	16	0.6	3.5	1.95	NHBP	0.9	0.7	0.2	19	16	106	-	-	✓	F	3.9	8/10	Well	Well
14.	28	36	57	Trace	150/90	Normal	20	0.6	3.3	1.85	NHBP	0.9	0.7	0.2	35	32	126	-	✓c	-	F	3.0	8/10	Well	Well
15.	22	37	59	Trace	140/100	Normal	18	0.6	3.7	2.15	NHBP	0.8	0.6	0.2	30	28	147	-	-	✓	F	2.9	8/10	Well	Well
16.	27	36	60	Trace	150/100	Normal	20	0.6	3.5	1.25	NHBP	0.8	0.5	0.3	28	24	186	✓	-	-	M	2.8	7/10	Well	Well
17.	32	28	64	Trace	140/100	Normal	22	0.6	3.7	2.05	NHBP	0.8	0.5	0.3	28	26	192	EAC	-	-	F	750 gm	IUD	Well	Dead Born

Sl. No.	Age	Gestational age (Weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg %	Sr. Creatinine mg%	Serum Uric acid mg%	Platelet Count Lakhs / mm³	Peripheral Smear	Sr. Bilirubin mg %			SG OT IU/L	SG PT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
												Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
18.	26	35	67	+++	150/90	Normal	20	0.6	4.1	1.05	NHBP	0.8	0.6	0.2	71	28	176	✓	-	-	M	1.8	8/10	Well	Well
19.	29	38	69	Trace	140/90	Normal	18	0.6	3.7	2.25	NHBP	0.9	0.6	0.3	30	32	162	✓	-	-	F	1.95	7/10	Well	Well
20.	27	37	59	Trace	130/100	Normal	19	0.6	3.3	1.25	NHBP	0.9	0.6	0.3	30	32	147	✓	-	-	M	2.5	IUD	Well	Dead Born
21.	26	32	62	Trace	140/100	Normal	18	0.6	3.5	2.45	NHBP	0.9	0.7	0.2	72	38	196	✓	-	-	F	1.4	8/10	Well	Well
22.	31	36	64	Trace	140/90	Normal	20	0.6	3.3	1.45	NHBP	0.8	0.6	0.2	22	24	118	✓c	-	-	M	2.7	8/10	Well	Well
23.	28	37	65	++	150/90	Normal	22	0.6	3.7	2.15	NHBP	0.8	0.5	0.3	20	24	126	-	-	✓	F	2.9	8/10	AbruptionWell	Well
24.	21	34	58	Trace	150/100	Normal	18	0.6	3.5	1.25	NHBP	0.9	0.7	0.2	22	26	114	✓	-	-	M	1.45	IUD	Well	Dead Born
25.	29	38	67	Trace	140/100	Normal	20	0.6	4.0	2.25	NHBP	0.8	0.6	0.2	28	26	124	✓	-	-	F	3.5	8/10	Well	Well
26.	32	37	56	Trace	130/100	Normal	18	0.6	4.1	2.35	NHBP	0.8	0.5	0.3	24	28	136	-	✓	-	F	3.25	9/10	Well	Well
27.	25	37	59	Trace	140/100	Normal	16	0.6	3.7	1.15	NHBP	0.9	0.6	0.3	30	24	134	-	-	✓	M	3.1	8/10	Well	Well
28.	23	28	57	Trace	140/90	Normal	20	0.6	3.3	1.95	NHBP	0.9	0.6	0.3	32	24	138	EAC	-	-	F	1.1	1/10	Well	Expired

SEVERE PRE ECLAMPSIA - PRIMI

Sl. No.	Age	Gestational age (Weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg%	Sr. Creatinine mg%	Serum Uric acid mg%	Platelet Count Lakhs / mm^3	Peripheral Smear	Sr. Bilirubin mg %			SGOT IU/L	SGPT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
												Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
1.	18	34	49	+	160/120	Normal	30	0.7	7.3	1.05	NHBP	0.9	0.6	0.3	33	31	191	-	-	✓C	F	1.75	8/10	Well	Well
2.	30	28	57	Trace	170/110	Normal	28	0.6	6.8	1.95	NHBP	0.9	0.6	0.3	81	87	182	EAC	-	-	M	850 gm	IUD	Well	Dead Born
3.	25	38	56	+++	170/120	Normal	28	0.7	8.2	1.25	NHBP	1.0	0.7	0.3	30	28	171	-	-	✓C	M	3.0	8/10	Well	Well
4.	21	30	54	++	180/110	Normal	32	0.7	8.5	1.15	NHBP	0.8	0.6	0.2	26	28	162	EAC	-	-	F	1.4	8/10	Well	Well
5.	26	32	60	+++	160/110	Grade I	20	0.6	6.2	1.05	Schistocytes+	0.8	0.6	0.2	116	118	672	✓C	-	-	F	1.7	7/10	HIE Well	Well
6.	27	37	62	+	160/120	Normal	30	0.6	9.0	1.85	NHBP	0.9	0.7	0.2	24	26	173	-	-	✓	M	2.7	8/10	Well	Well
7.	19	28	50	+++	160/120	Normal	26	0.6	7.8	1.1	NHBP	0.9	0.6	0.2	28	28	167	EAC	-	-	F	950 gm	IUD	Well	Dead Born
8.	22	39	61	+	160/110	Normal	26	0.7	8.2	1.25	NHBP	0.8	0.5	0.3	22	26	156	-	-	✓	F	3.5	8/10	Well	Well
9.	31	32	56	++	170/110	Normal	28	0.6	8.4	1.95	NHBP	0.8	0.5	0.3	30	32	184	EAC	-	-	M	1.45	8/10	Well	Well
10.	23	34	66	++++	170/110	Grade I	31	0.9	8.6	80,000	Schistocytes+	2.3	1.5	0.8	261	265	684	-	✓	-	F	2.0	IUD	ARF Expired	Dead Born
11.	26	40	62	+++	170/120	Normal	28	0.8	8.4	1.2	NHBP	0.9	0.7	0.2	39	36	192	-	-	✓	M	3.7	8/10	Well	Well
12.	24	34	49	+	160/110	Normal	26	0.6	7.8	1.75	NHBP	0.9	0.6	0.3	24	22	116	✓	-	-	M	1.5	IUD	Well	Dead Born
13.	32	28	61	++	160/110	Normal	24	0.6	7.6	1.15	NHBP	1.0	0.7	0.3	24	24	124	EAC	-	-	F	750	IUD	Abruption	Dead

Sl. No.	Age	Gestational age (Weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg%	Sr. Creatinine mg%	Serum Uric acid mg%	Platelet Count Lakhs / mm³	Peripheral Smear	Sr. Bilirubin mg %			SGOT IU/L	SGPT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
												Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
																						gm		Well	Born
14.	29	36	48	Trace	170/110	Grade I	28	0.6	7.7	2.25	NHBP	0.8	0.5	0.3	20	26	116	-	-	✓TWINS	F F	1.6 1.7	8/10	Well	Well
15.	17	38	45	+	170/110	Normal	30	0.7	7.5	1.05	NHBP	0.8	0.6	0.2	22	20	125	✓c	-	-	M	2.6	8/10	Well	Well
16.	28	34	63	+++	170/120	Normal	30	0.6	7.3	2.15	NHBP	0.9	0.7	0.2	96	94	612	✓c	-	-	F	1.7	8/10	Well	Well
17.	23	36	55	++++	180/120	Grade I	28	0.6	7.5	2.05	NHBP	0.8	0.5	0.3	24	26	192	✓	-	-	M	1.6	3/10	ARF Well	Expired
18.	25	36	64	+	160/110	Normal	28	0.6	7.1	1.35	NHBP	0.9	0.6	0.3	22	28	186	✓c	-	-	F	1.8	8/10	Well	Well
19.	31	32	56	++	160/110	Normal	26	0.6	8.0	1.4	NHBP	0.9	0.6	0.3	30	32	148	✓	-	-	F	1.40	8/10	Well	Well
20.	26	34	54	+	170/110	Normal	28	0.7	7.2	1.25	NHBP	0.9	0.7	0.2	32	30	168	✓	-	-	M	1.9	IUD	Peripartum Cardiomyopath, GSH	Dead Born
21.	17	38	60	+++	170/120	Normal	30	0.7	7.6	1.95	NHBP	0.9	0.6	0.3	82	86	184	-	-	✓	F	1.9	8/10	Abruption Well	Well
22.	20	37	46	+	160/110	Normal	26	0.6	8.2	1.15	NHBP	1.0	0.8	0.2	30	28	172	✓c	-	-	M	2.2	8/10	Well	Well
23.	27	28	62	++++	180/120	Grade I	24	0.6	7.3	1.3	NHBP	0.8	0.6	0.2	26	28	168	EAC	-	-	M	600 gm	IUD	Well	Dead Born
24.	22	30	49	++	160/110	Normal	30	0.6	8.8	1.85	NHBP	0.8	0.5	0.3	24	22	146	✓	-	-	F	1.2	8/10	Well	Well
25.	19	36	45	+	160/110	Normal	32	0.7	7.4	1.15	NHBP	0.9	0.7	0.2	20	24	172	-	-	✓	F	1.8	8/10	Well	Well

Sl. No.	Age	Gestational age (Weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg%	Sr. Creatinine mg%	Serum Uric acid mg%	Platelet Count Lakhs / mm³	Peripheral Smear	Sr. Bilirubin mg %			SGOT IU/L	SGPT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
												Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
26.	28	32	61	+++	170/110	Normal	28	0.6	9.0	2.25	NHBP	0.8	0.5	0.3	28	30	184	✓	-	-	M	1.0	3/10	Well	Expired
27.	30	37	54	+	160/120	Grade I	26	0.6	9.2	1.35	NHBP	0.9	0.6	0.3	28	28	176	✓c	-	-	F	2.5	8/10	Well	Well
28.	24	34	60	++	160/110	Normal	32	0.7	8.0	1.25	NHBP	0.9	0.6	0.3	24	22	124	✓	-	-	M	1.7	8/10	Well	Well
29.	25	34	51	++	150/110	Normal	30	1.0	7.8	1.05	Fragmented RBC's	0.8	0.6	0.2	362	381	674	-	-	LSCS Hysterectomy	M	1.15	8/10	Abruption ARF Expired	Well
30.	26	38	62	+	150/110	Normal	28	0.6	8.2	1.75	NHBP	0.9	0.7	0.2	22	20	116	-	-	✓	M	3.1	7/10	Well	Well
31.	22	34	51	++	160/110	Normal	28	0.7	7.8	1.05	NHBP	0.9	0.6	0.3	24	22	120	✓c	-	-	F	1.5	3/10	Well	Expired
32.	27	39	64	+	170/110	Normal	26	0.6	7.6	1.85	NHBP	1.0	0.7	0.3	30	28	162	-	-	✓	F	2.7	6/10	Well	Well
33.	31	28	61	+	160/110	Normal	30	0.8	7.3	1.35	NHBP	0.8	0.5	0.3	28	30	172	EAC	-	-	F	700 gm	IUD	Well	Dead Born
34.	21	40	49	+++	170/110	Grade I	24	0.6	8.0	1.95	NHBP	0.9	0.6	0.3	30	32	184	-	-	✓c	M	3.2	6/10	Well	Well
35.	28	32	60	++	170/110	Normal	22	0.6	7.1	1.95	NHBP	1.0	0.8	0.2	30	28	176	✓	-	-	F	1.5	IUD	Well	Dead Born
36.	21	40	47	+	150/120	Normal	24	0.7	9.0	1.05	NHBP	0.8	0.5	0.3	28	30	124	✓c	-	-	M	3.1	8/10	Well	Well
37.	18	PN	51	+++	150/120	Grade II	23	0.7	6.8	1.15	Burcells	0.9	0.6	0.3	152	148	668	✓ Outside	-	-	F	2.1	8/10	Well	Well
38.	25	36	52	+++	160/110	Normal	28	0.6	8.2	2.05	NHBP	0.8	0.5	0.3	22	24	118	✓c	-	-	M	2.2	8/10	Well	Well

Sl. No.	Age	Gestational age (Weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg%	Sr. Creatinine mg%	Serum Uric acid mg%	Platelet Count Lakhs / mm³	Peripheral Smear	Sr. Bilirubin mg %			SGOT IU/L	SGPT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
												Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
39.	31	36	66	+++	170/110	Normal	28	0.7	7.3	1.15	NHBP	0.9	0.6	0.3	36	34	194	-	-	✓	M	1.9	8/10	Well	Well
40.	22	34	54	++	170/120	Normal	27	0.6	7.6	1.95	NHBP	0.8	0.6	0.2	33	32	176	✓	-	-	F	1.75	8/10	Well	Well
41.	25	37	63	+++	160/110	Normal	28	0.6	7.8	1.35	NHBP	0.9	0.6	0.3	20	28	107	✓c	-	-	M	2.4	7/10	Well	Well
42.	26	39	60	+	160/110	Normal	24	0.6	6.8	1.85	NHBP	1.0	0.7	0.3	22	24	126	-	-	✓c	F	2.8	7/10	Well	Well
43.	27	34	61	++	160/110	Normal	30	0.7	9.0	1.75	NHBP	0.9	0.7	0.2	28	30	132	✓c	-	-	F	1.6	8/10	Well	Well
44.	23	36	49	+++	170/120	Normal	28	0.6	8.0	1.35	NHBP	0.8	0.5	0.3	26	28	148	✓	-	-	M	1.8	8/10	Well	Well
45.	21	28	58	++++	170/120	Grade II	22	0.7	7.1	70,000	Schistocytes+	0.9	0.6	0.3	182	186	692	EAC	-	-	M	800 gm	IUD	Well	Dead Born
46.	24	38	48	+	160/110	Normal	26	0.6	8.2	1.45	NHBP	0.8	0.6	0.2	30	26	126	✓c	-	-	F	2.6	8/10	Well	Well
47.	26	37	62	++	150/120	Normal	30	0.7	8.6	2.25	NHBP	0.9	0.7	0.2	28	28	138	-	-	✓	M	2.5	7/10	Well	Well
48.	30	32	64	+	150/110	Normal	30	0.8	6.7	1.25	NHBP	0.9	0.6	0.3	24	26	124	✓	-	-	F	1.1	3/10	Abruption Well	Expired
49.	24	38	60	++	160/110	Normal	28	0.6	6.9	1.65	NHBP	0.8	0.6	0.2	32	30	136	-	-	✓	M	2.7	8/10	Well	Well
50.	28	37	61	+++	170/120	Normal	32	0.8	7.9	1.65	NHBP	0.8	0.5	0.3	91	93	212	-	-	✓c	F	2.8	8/10	Well	Well
51.	18	38	47	++	180/110	Normal	26	0.6	8.8	1.35	NHBP	0.9	0.6	0.3	30	24	186	✓c	-	-	F	2.2	8/10	Well	Well
52.	29	36	49	++	160/110	Normal	24	0.6	9.0	1.15	NHBP	0.9	0.6	0.3	28	26	124	-	-	✓	M	1.8	8/10	Well	Well

SEVERE PREECLAMPSIA - MULTI

Sl. No.	Age	Gestational age (Weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg %	Sr. Creatinine mg%	Serum Uric acid mg%	Platelet Count Lakhs / mm³	Peripheral Smear	Sr. Bilirubin mg %			SG OT IU/L	SG PT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
												Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
1.	25	30	60	+++	160/120	Grade I	22	0.6	7.1	1.15	NHBP	1.0	0.7	0.3	22	26	98	✓	-	-	F	1.25	8/10	Well	Well
2.	30	32	62	+	150/110	Normal	20	0.6	6.7	1.65	NHBP	0.9	0.7	0.2	20	22	96	-	-	✓c	M	1.4	8/10	Well	Well
3.	21	38	49	++	160/110	Normal	30	0.7	6.3	1.25	NHBP	0.9	0.6	0.3	28	30	126	✓c	-	-	F	2.1	8/10	Well	Well
4.	27	34	49	+	150/120	Normal	28	0.6	7.3	1.85	NHBP	0.9	0.6	0.3	32	30	184	✓	-	-	F	1.6	8/10	Well	Well
5.	23	38	60	++	160/110	Normal	26	0.6	7.8	1.05	NHBP	1.0	0.7	0.3	28	26	166	-	-	✓c	M	2.9	7/10	Well	Well
6.	19	37	62	+++	170/110	Normal	28	0.6	8.2	1.05	Schistocytes+	0.9	0.6	0.3	94	90	611	✓c	-	-	F	1.8	8/10	Well	Well
7.	22	36	47	++	160/110	Normal	24	0.6	8.0	2.25	NHBP	0.9	0.6	0.3	24	28	162	✓	-	-	F	1.9	8/10	Well	Well
8.	26	28	63	+	150/110	Normal	28	0.7	8.1	1.15	NHBP	0.8	0.6	0.2	26	24	126	✓	-	-	M	1.0	IUD	Well	Dead Born
9.	31	38	54	+++	160/120	Grade I	30	0.7	7.3	1.95	NHBP	0.8	0.5	0.3	28	24	118	-	-	✓	M	3.1	2/10	Abruption Well	Expired
10.	24	32	56	+	160/110	Normal	22	0.6	7.1	1.35	NHBP	1.0	0.6	0.4	30	28	114	✓	-	-	F	1.3	8/10	Well	Well
11.	28	24	66	+	150/110	Normal	24	0.6	7.0	1.85	NHBP	0.9	0.6	0.3	30	30	126	EAC	-	-	M	550 gm	IUD	Well	Dead Born
12.	22	36	50	+++	170/110	Normal	20	0.6	7.0	1.25	NHBP	0.9	0.6	0.3	28	26	138	✓	-	-	F	1.8	8/10	Well	Well
13.	24	30	68	++	160/110	Normal	22	0.6	7.8	1.15	Schistocytes+	0.8	0.5	0.3	86	88	608	✓	-	-	F	2.0	IUD	Well	Dead Born
14.	23	37	49	++	170/110	Normal	18	0.6	6.7	1.35	NHBP	0.9	0.7	0.2	30	32	124	-	-	✓	F	1.9	7/10	Abruption Well	Well
15.	25	28	57	+	160/110	Normal	20	0.6	7.0	1.75	NHBP	0.9	0.6	0.3	34	32	132	EAC	-	-	M	1.1	IUD	Well	Dead Born
16.	33	38	67	+	150/110	Normal	28	0.6	6.8	1.45	NHBP	0.8	0.6	0.2	30	28	146	-	-	✓	M	3.9	8/10	Well	Well

Sl. No.	Age	Gestational age (Weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg %	Sr. Creatinine mg%	Serum Uric acid mg%	Platelet Count Lakhs / mm³	Peripheral Smear	Sr. Bilirubin mg %			SG OT IU/L	SG PT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
												Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
17.	21	30	49	Trace	150/110	Grade I	18	0.6	7.1	1.65	NHBP	0.8	0.5	0.3	26	24	138	✓	-	-	F	950 gm	8/10	Well	Well
18.	29	40	61	+++	160/120	Normal	26	0.6	6.3	1.3	NHBP	0.8	0.5	0.3	28	26	162	✓	-	-	F	2.0	2/10	Well	Expired
19.	20	34	47	+++	170/110	Normal	30	0.7	6.8	1.1	NHBP	0.9	0.6	0.3	30	28	184	✓c	-	-	M	1.75	8/10	Well	Well
20.	32	34	56	++	150/110	Normal	20	0.6	6.1	1.15	NHBP	0.9	0.7	0.2	32	30	192	✓	-	-	M	1.75	8/10	Well	Well
21.	23	36	50	+	160/110	Normal	28	0.6	6.3	1.85	NHBP	0.9	0.6	0.3	28	32	186	-	-	✓	F	1.8	8/10	Well	Well
22.	28	32	52	++	150/110	Normal	23	0.6	7.6	1.2	Fragmented RBC's	0.8	0.6	0.2	124	128	652	✓	-	-	M	1.4	2/10	Well	Expired
23.	22	37	51	++	160/120	Normal	26	0.6	6.7	1.35	NHBP	0.8	0.6	0.2	28	26	126	-	-	✓	F	2.7	8/10	Well	Well
24.	27	38	62	+	170/110	Normal	30	0.7	6.8	1.95	NHBP	1.0	0.7	0.3	24	22	134	✓	-	-	M	2.8	8/10	Well	Well
25.	34	28	55	+	160/110	Normal	28	0.6	7.0	1.45	NHBP	1.0	0.6	0.4	22	26	148	EAC	-	-	F	900 gm	6/10	Well	Well
26.	24	40	49	+++	180/120	Normal	32	0.7	6.7	2.05	NHBP	0.9	0.6	0.3	26	28	162	-	-	✓	M	2.9	3/10	Well	Expired
27.	26	37	62	+	170/110	Normal	30	0.6	8.0	2.25	NHBP	0.8	0.6	0.2	30	26	156	✓	-	-	M	1.9	7/10	Well	Well
28.	31	38	59	++	160/120	Normal	24	0.7	7.3	1.05	Burrcells	0.9	0.5	0.4	81	84	632	✓ TWINS	-	-	F F	1.8 1.9	8/10 8/10	Well	Well
29.	28	36	63	++	170/110	Normal	28	0.6	6.8	1.95	NHBP	0.9	0.7	0.2	32	30	106	✓c	-	-	M	2.5	8/10	Well	Well
30.	20	32	50	+	160/110	Normal	24	0.6	7.1	1.25	NHBP	0.8	0.5	0.3	30	28	128	✓	-	-	F	1.35	8/10	Well	Well
31.	30	38	62	+++	170/120	Grade I	26	0.6	7.0	1.65	NHBP	0.9	0.6	0.3	26	28	134	✓	-	-	F	2.6	7/10	Well	Well
32.	22	34	49	+	160/110	Normal	20	0.6	8.0	1.05	NHBP	0.9	0.7	0.2	24	26	126	✓c	-	-	F	1.9	8/10	Well	Well
33.	19	38	47	+	150/110	Normal	18	0.6	8.1	1.65	NHBP	0.8	0.5	0.3	28	24	148	-	-	✓	M	2.9	7/10	Well	Well

Sl. No.	Age	Gestational age (Weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg %	Sr. Creatinine mg%	Serum Uric acid mg%	Platelet Count Lakhs / mm³	Peripheral Smear	Sr. Bilirubin mg %			SG OT IU/L	SG PT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
												Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
34.	23	28	55	+++	150/120	Grade II	20	0.7	7.3	1.35	NHBP	0.8	0.6	0.2	30	28	154	EAC	-	-	M	1.0	IUD	Well	Dead Born
35.	26	37	67	++	160/110	Normal	24	0.6	7.1	1.75	NHBP	0.9	0.6	0.3	32	28	128	✓	-	-	M	3.1	8/10	Well	Well
36.	23	34	49	+++	170/110	Normal	26	0.6	7.2	1.1	Schistocytes+	0.9	0.6	0.3	84	86	626	✓	-	-	F	2.2	IUD	Well	Dead Born
37.	21	36	59	++	170/110	Normal	26	0.6	6.7	1.85	NHBP	0.8	0.6	0.2	22	26	164	-	-	✓	M	1.95	8/10	Well	Well
38.	28	30	68	+	160/110	Normal	28	0.7	6.8	1.65	NHBP	0.9	0.6	0.3	28	24	132	EAC	-	-	M	900 gm	IUD	Well	Dead Born
39.	31	34	54	+++	170/110	Normal	24	0.6	6.8	1.25	NHBP	0.9	0.6	0.3	34	32	124	✓c	-	-	F	1.7	8/10	Well	Well
40.	27	28	56	+	160/110	Normal	18	0.6	6.7	2.15	NHBP	0.8	0.6	0.2	28	26	166	EAC	-	-	M	1.0	IUD	Well	Dead Born
41.	24	37	63	+	150/110	Normal	22	0.6	6.1	1.15	NHBP	0.9	0.7	0.2	24	28	184	-	-	✓	M	3.2	7/10	Well	Well
42.	28	32	60	++	160/110	Normal	30	0.7	7.1	1.95	NHBP	1.0	0.7	0.3	30	28	128	-	-	✓	M	1.45	IUD	Well	Dead Born
43.	27	36	54	++	160/120	Normal	19	0.6	6.9	1.15	Schistocytes+	0.8	0.5	0.3	86	84	624	✓	-	-	F	1.8	2/10	Well	Expired
44.	25	38	54	+++	170/120	Grade I	28	0.6	6.8	1.75	NHBP	1.0	0.7	0.3	20	28	176	-	✓	-	M	3.3	7/10	Well	Well
45.	32	30	64	+	160/110	Normal	26	0.6	7.1	1.35	NHBP	0.9	0.6	0.3	30	26	168	EAC	-	-	F	1.2	IUD	Well	Dead Born
46.	23	39	51	+	150/110	Normal	20	0.6	7.3	1.65	NHBP	0.8	0.5	0.3	32	30	144	✓	-	-	F	2.7	7/10	Well	Well
47.	29	32	62	++	160/120	Normal	18	0.7	6.5	1.85	NHBP	0.9	0.6	0.3	102	110	622	✓	-	-	M	1.4	2/10	Well	Expired
48.	33	37	54	Trace	170/110	Normal	32	0.7	8.0	1.05	NHBP	0.8	0.6	0.2	19	35	104	✓	-	-	F	3.1	8/10	Well	Well

ECLAMPSIA

Sl. No.	Type of Eclampsia	Age	Gravida	Gestational age (Weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg %	Sr. Creatinine mg%	Serum Uric acid mg%	Platelet Count Lakhs / mm^3	Peripheral Smear	Sr. Bilirubin mg %			SGOT IU/L	SGPT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
														Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
1.	AP	21	P	34	51	+	140/90	Normal	22	0.6	6.2	1.15	NHBP	0.9	0.6	0.3	32	36	194	-	-	✓	F	1.95	7/10	Well	Well
2.	AP	18	P	28	56	++++	150/100	Normal	18	0.7	6.4	1.85	NHBP	0.9	0.7	0.2	75	78	604	EAC	-	-	M	750 gm	IUD	Well	Dead Born
3.	AP	32	M	32	48	Trace	160/110	Normal	20	0.6	6.3	1.75	NHBP	0.8	0.6	0.2	26	24	112	✓	-	-	M	1.9	7/10	Well	Well
4.	AP	26	M	30	49	++	160/100	Normal	22	0.6	6.7	1.45	NHBP	0.8	0.5	0.3	24	28	142	EAC	-	-	F	1.4	7/10	Cerebral Edema Well	Well
5.	AP	27	M	36	60	+	150/110	Normal	19	0.6	6.3	1.25	NHBP	0.9	0.6	0.3	22	26	146	-	-	✓	F	2.7	7/10	Well	Well
6.	AP	20	P	34	48	+++	140/110	Grade I	24	0.9	8.2	75,000	Burr cells	0.9	0.6	0.3	254	256	686	-	-	✓c	F	1.8	IUD	Abruption CRA Expired	Dead Born
7.	AP	24	M	28	66	+++	150/100	Normal	22	0.6	7.1	1.65	NHBP	0.8	0.5	0.3	24	24	162	EAC	-	-	M	850 gm	1/10	Well	Expired
8.	PP	25	M	36	58	+	140/110	Normal	20	0.7	8.0	1.75	NHBP	0.8	0.6	0.2	20	22	156	✓c	-	-	M	3.5	8/10	Well	Well
9.	AP	19	P	38	49	+	160/120	Normal	19	0.6	8.1	1.05	NHBP	0.9	0.6	0.3	21	23	164	-	-	✓	M	3.6	7/10	Cerebral Edema GSH Well	Well
10.	AP	31	M	39	60	+	160/110	Normal	18	0.7	7.2	1.85	NHBP	1.0	0.7	0.3	81	80	256	-	-	✓	F	2.7	8/10	Well	Well
11.	PP	25	P	38	61	++	170/110	Grade II	20	0.6	7.4	1.35	NHBP	0.9	0.6	0.3	18	16	171	✓	-	-	F	2.8	8/10	Well	Well
12.	AP	22	M	38	49	++	150/110	Normal	22	0.8	7.6	1.15	Schistocytes+	0.9	0.6	0.3	82	86	648	✓	-	-	M	2.6	8/10	Well	Well
13.	IP	26	P	40	58	+	150/100	Normal	24	0.6	6.4	1.7	NHBP	0.9	0.5	0.4	18	18	91	-	-	✓c	F	2.6	8/10	Abruption Well	Well

Sl. No.	Type of Eclampsia	Age	Gravida	Gestational age (Weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg %	Sr. Creatinine mg%	Serum Uric acid mg%	Platelet Count Lakhs / mm^3	Peripheral Smear	Sr. Bilirubin mg %			SGOT IU/L	SGPT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
														Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
14.	AP	30	M	28	59	++	160/100	Normal	26	0.7	6.3	1.25	NHBP	0.8	0.5	0.3	19	18	96	EAC	-	-	M	1.1	2/10	HIE GSH	Expired
15.	AP	27	M	30	57	Trace	170/100	Normal	22	0.6	6.0	1.4	NHBP	0.9	0.6	0.3	22	24	101	✓	-	-	F	1.3	1/10	Well	Expired
16.	AP	19	P	32	48	+	140/110	Grade I	20	0.6	7.1	1.8	NHBP	0.8	0.6	0.2	20	19	112	-	-	✓	M	1.6	IUD	Well	Dead Born
17.	AP	22	P	38	49	+++	140/100	Normal	20	0.6	6.1	2.25	NHBP	0.9	0.6	0.3	74	48	192	-	-	✓	M	2.6	8/10	Well	Well
18.	AP	24	M	40	58	+	140/100	Normal	18	0.6	6.0	2.15	NHBP	0.8	0.6	0.2	26	24	106	-	-	✓	F	3.0	8/10	Well	Well
19.	AP	23	P	34	56	+++	150/110	Normal	22	0.7	6.4	1.15	NHBP	0.9	0.7	0.2	18	19	112	-	-	✓	F	2.7	8/10	Well	Well
20.	PP	23	M	32	60	+	150/100	Normal	20	0.6	6.7	1.85	NHBP	0.9	0.6	0.3	16	18	122	✓	-	-	M	1.75	7/10	Well	Well
21.	AP	17	P	40	47	+	140/110	Normal	24	0.7	6.4	1.75	NHBP	1.0	0.7	0.3	20	22	116	-	-	✓	F	3.5	9/10	Well	Well
22.	AP	20	P	37	51	++	160/110	Grade II	19	0.6	7.0	1.25	NHBP	0.8	0.6	0.2	16	16	104	-	-	✓	F	2.9	9/10	Well	Well
23.	IP	24	M	34	62	++++	160/100	Normal	20	0.6	7.1	1.65	NHBP	0.8	0.5	0.3	19	17	102	-	✓	-	F	1.9	8/10	Well	Well
24.	AP	21	P	38	50	+	160/100	Normal	24	0.6	6.7	1.8	NHBP	0.9	0.7	0.2	72	42	110	-	-	✓	M	2.5	IUD	Well	Dead Born
25.	PP	20	P	36	46	+++	140/100	Normal	20	0.6	6.8	1.15	NHBP	0.8	0.6	0.2	18	22	126	-	-	✓	F	3.1	9/10	Well	Well
26.	AP	31	M	28	59	++	150/100	Normal	18	0.6	7.0	1.15	Burr cells	0.9	0.6	0.3	92	96	632	-	-	✓	M	1.6	1/10	Cerebral Edema Well	Expired
27.	AP	27	P	30	49	+	140/110	Grade I	22	0.7	7.1	1.25	NHBP	0.9	0.6	0.3	20	21	140	✓	-	-	F	1.4	2/10	Well	Expired
28.	AP	28	M	39	63	+	160/100	Normal	24	0.6	6.8	1.95	NHBP	0.8	0.6	0.2	24	22	134	✓c	-	-	M	2.4	1/10	Well	Expired
29.	PP	26	P	40	60	++	170/100	Normal	17	0.6	6.7	2.25	NHBP	0.9	0.7	0.2	74	77	607	✓	-	-	F	3.0	8/10	Well	Well

Sl. No.	Type of Eclampsia	Age	Gravida	Gestational age (Weeks)	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg %	Sr. Creatinine mg%	Serum Uric acid mg%	Platelet Count Lakhs / mm³	Peripheral Smear	Sr. Bilirubin mg %			SGOT IU/L	SGPT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
														Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
30.	AP	27	P	32	61	+	160/110	Normal	20	0.6	7.2	1.25	NHBP	0.8	0.5	0.3	18	20	118	-	-	✓	M	1.8	IUD	Well	Dead Born
31.	IP	32	M	34	63	+++	140/100	Normal	22	0.7	7.0	1.4	NHBP	0.9	0.6	0.3	19	22	96	-	-	✓	F	2.1	8/10	Cerebral Edema Well	Well
32.	AP	23	P	38	58	++	150/100	Normal	18	0.6	6.0	2.2	NHBP	0.8	0.5	0.3	24	26	102	-	-	✓	M	3.75	8/10	Well	Well

HELLP SYNDROME

Sl. No.	Group	Age	Gravida	Gestational age	Wt. Kg	Urine Alb.	BP mm-Hg	Fundus	Blood Urea mg %	Sr. Creatinine mg %	Serum Uric acid mg%	Platelet Count Lakhs / mm³	Peripheral Smear	Sr. Bilirubin mg %			SGOT IU/L	SGPT IU/L	Sr. LDH IU / L	Mode of delivery			Baby			Maternal Outcome	Fetal Outcome
														Total	Direct	Indirect				L.N.	Forceps	LSCS	Sex	Wt kg	Apg 1 mt		
1.	Mild PE	23	M	39	56	+	140/90	Normal	19	0.6	6.8	1.2	Schistocytes+	0.8	0.5	0.3	76	78	611	-	-	✓	F	3.1	8/10	Well	Well
2.	Severe PE	26	P	32	60	+++	160/110	Grade I	20	0.6	6.2	1.05	Schistocytes+	0.8	0.6	0.2	116	118	672	✓c	-	-	F	1.7	7/10	HIE Well	Well
3.	Severe PE	23	P	34	66	++++	170/110	Grade I	31	0.9	8.6	80,000	Schistocytes+	2.3	1.5	0.8	261	265	684	-	✓	-	F	2.0	IUD	ARF Expired	Dead Born
4.	Severe PE	25	P	34	57	++	150/110	Normal	30	1.0	7.8	1.05	Fragmented RBC's	0.8	0.6	0.2	362	381	674	-	-	LSCS Hysterectomy	M	1.15	8/10	Abruptiion ARF Expired	Well
5.	Severe PE	18	P	PN	51	+++	150/120	Grade II	23	0.7	6.8	1.15	Burr cells	0.9	0.6	0.3	152	148	668	✓ outside	-	-	F	2.1	8/10	Well	Well
6.	Severe PE	21	P	28	58	++++	170/120	Grade II	22	0.7	7.1	70,000	Schistocytes+	0.9	0.6	0.3	182	186	692	EAC	-	-	M	800 gm	IUD	Well	Dead Born
7.	Severe PE	19	M	37	62	+++	170/110	Normal	18	0.6	8.2	1.05	Schistocytes+	0.9	0.6	0.3	94	90	611	✓C	-	-	F	1.8	8/10	Well	Well
8.	Severe PE	24	M	30	68	++	160/110	Normal	22	0.6	7.8	1.15	Schistocytes+	0.8	0.5	0.3	86	88	608	✓	-	-	F	2.0	IUD	Well	Dead Born
9.	Severe PE	28	M	32	62	++	150/110	Normal	23	0.6	7.6	1.2	Fragmented RBC's	0.8	0.6	0.2	124	128	652	✓	-	-	M	1.4	2/10	Well	Expired
10.	Severe PE	31	M	38	59	++	160/120	Normal	24	0.7	7.3	1.05	Burr cells	0.9	0.5	0.4	81	84	632	✓ Twins	-	-	F F	1.8 1.9	8/10 8/10	Well	Well
11.	Severe PE	23	M	34	49	+++	170/110	Normal	26	0.6	7.2	1.1	Schistocytes+	0.9	0.6	0.3	84	86	626	✓	-	-	F	2.2	IUD	Well	Dead Born

12.	Severe PE	27	M	36	54	++	160/120	Normal	19	0.6	6.9	1.15	Schistocytes+	0.8	0.5	0.3	86	84	624	✓	-	-	F	1.8	2/10	Well	Expired
13.	AP Eclampsia	20	P	34	48	+++	140/110	Grade I	24	0.9	8.2	75,000	Burr Cells	0.9	0.6	0.3	254	256	686	-	-	✓c	F	1.8	IUD	Abruption CRA Expired	Dead Born
14.	AP Eclampsia	22	M	38	49	++	150/110	Normal	22	0.8	7.6	1.15	Schistocytes+	0.9	0.6	0.3	82	86	648	✓	-	-	M	2.6	8/10	Well	Well
15.	AP Eclampsia	31	M	28	59	++	150/100	Normal	18	0.6	7.0	1.15	Burr Cells	0.9	0.6	0.3	92	96	632	-	-	✓	M	1.6	1/10	Cerebral Edema Well	Expired